Evidence of Health Effects of Electromagnetic Radiation, To the Australian Senate Inquiry into Electromagnetic Radiation

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Abstract:

Genotoxic and epidemiological evidence was presented to the Australian Senate Inquiry. The Inquiry Chairperson, Senator Lyn Allison, described Dr Neil Cherry's evidence as the only independent professional evidence not related to industry. Since he spent most of June and July that year in Europe presenting evidence in Italy, Austria, Ireland, Switzerland and the European Parliament in Brussels, and collecting research results from the other presentations of world leading independent researchers, the evidence presented here was strongly updated. The conclusions from this evidence are strongly contrasted with the position of Dr Michael Repacholi from the WHO, ICNIRP (International Commission on Non-Ionizing Radiation Protection), the Australian Radiation Laboratory and many other "authorities" around the world.

Introduction:

I will show you evidence that I assess, using classic public health assessment methods, classifies electromagnetic radiation as causally associated with cancer, reproductive, neurological and cardiac illness and death. The dose-response relationships identify a consistent threshold, showing that the safe exposure level of zero exposure. This is expected from neurologically and cellular resonantly interactive substances and genotoxic substances.

This issue has been so politicized. There are two major casualties, the truth and public health.

We have natural EMR-based communication systems in our brains, hearts, cell and bodies. External natural and artificial EMR resonantly interacts with these communication systems altering hormone balances and damaging organs and cells. The brain and the heart are especially sensitive because they mediate and regulate primary biological functions that are vital to life, thinking and heart beat, using EMR signals, the EEG and ECG. When EMR interferes with the EEG this is communicated to the body by neurotransmitters and neurohormones, including the serotonin/melatonin system. EMR reduces melatonin. Melatonin is vital for the health of the Immune System, the Brain, The Heart and every cell, because it is the most potent naturally produced antioxidant. It is a potent free radical scavenger that plays a vital protective role to protect the DNA in every cell. Reduced melatonin causes cancer, miscarriage, heart disease, neurological diseases, viral and bacterial diseases, etc…

Both through reducing melatonin and through enhancing free radical activity EMR is genotoxic, damaging the DNA and chromosomes, enhancing oncogene expression and transforming cells to neoplastic cells and causing cancer in exposed populations.

Intense chromosome damage of fetuses in the uterus by microwaves, causes prompt miscarriage. Less intense chromosome damage, largely but not totally repaired by the body's cell repair system, such as is caused by radio waves, causes fetal damage, congenital malformation, still birth, cot death, etc..

Neurological effects, such as headache, sleep disturbance, concentration disturbance, short-term memory loss, dizziness and nausea, are acute effects that can be experienced over minutes, hours, days and months. However, cell damage in the brain causes significantly accelerated cell death. Over years this means long-term memory loss, and neurological diseases such as Epilepsy, Parkinson's Disease, Multiple Sclerosis, Alzheimer's Disease, and sudden death from violent epileptic seizures.

The heart is similar with short-term problems with cardiac arrhythmia, missed heart beats, etc, sudden death from Heart Attacks and chronic illness and death from heart disease, especially arrhythmic diseases.

Cancer is a chronic disease problem from accumulated genetic cell damage. Latencies for children and soft tissue cancers are as short as a few years, for most cancers they take 10 to 40 years to develop. Cancer rates rise rapidly with age over 65 years because of the life-time of accumulated cell damage and the drastic reduction in melatonin that occurs after puberty.

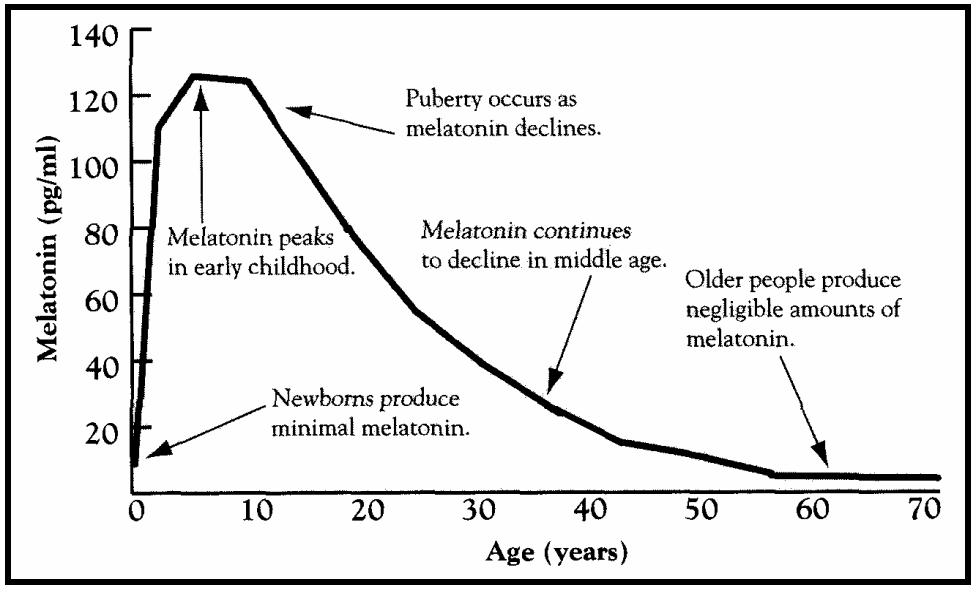

Figure 1: Melatonin production varies with age, Reiter and Robinson (1995).

This shows how vulnerable very young children are because they have very low melatonin levels and undeveloped immune systems. It also shows how reduced melatonin makes older people more vulnerable and much more prone to disease and cancer.

This also illustrates how useful and important it is to know about the natural systems when assessing health effects. Our bodies have developed strong natural protection and repair mechanisms. Many chemicals interfere with these natural systems and are therefore seen

as toxic and dangerous. Xenoestrogens, such as organochlorines, act on the estrogen receptors on cells, especially breast cells, and, through altering the estrogen levels, enhance the occurrence of breast cancer. Melatonin reduction is similar because it is a neurohormone with receptors in cells and nuclei to control many cellular processes, including in breast cells.

A large epidemiological study of female breast cancer over 24 States in the U.S., Cantor et al. (1995), identified several organic solvents, including organochlorines, that significantly increased the incidence of breast cancer, Table 1.

Table 1: Breast cancer from occupational exposures, Cantor et al. (1995)

Substance	Odds Ratio	95%Confidence Interval
Carbon Tetrachloride	1.13	1.1 - 1.2
Methylene chloride	1.15	1.1 - 1.2
Styrene	1.18	1.1 - 1.3
Metals and Oxides	1.13	1.0 - 1.3
Ionizing Radiation	1.14	0.9 - 1.4
Radiofrequency fields	1.15	1.1 - 1.2

Radiofrequency fields are as dangerous as toxic chemicals and Ionizing Radiation.

This is backed by over 10 other studies showing that EMR across the spectrum increases breast cancer incidence and 15 studies showing reduced melatonin, including three with dose-response relationships. These are sufficient to classify a causal relationship between EMR and breast cancer, with melatonin reduction is the biological mechanism.

This conclusion and this evidence is strongly contrasted with the position of Dr Michael Repacholi from the WHO, ICNIRP (International Commission on Non-Ionizing Radiation Protection), the Australian Radiation Laboratory (whatever it is called now), and many other "authorities" around the world.

This is because my background and my approach is very different from the "established authorities" nationally and internationally. This is also why I have been in strong demand internationally to explain this situation and to present what the scientific research actually shows, in the context of public health protection.

Professional Background and Experience:

As a meteorologist and biophysicist I have worked for over 20 years as an Agricultural Meteorologist. I took my B.Sc. (Hons) and Ph.D. in Physics, in solid state physics and atmospheric physics, respectively. At McGill University I researched and taught about the meteorology of thunderstorms and air pollution. I continued this at Auckland University and added wind energy.

I was then appointed the lecturer in Agricultural Meteorology at Lincoln University. This took me into environmental physics and biophysics as I researched the effects of weather and climate on plants, animals and people. This included aspects of plant, animal and

human physiology and environmental epidemiology. I led research and teaching in three areas over a period of 20 years. This included wind and solar energy; climatology, climate change and seasonal weather forecasting; and human biometeorology. The statistical methods of climatology and environmental epidemiology are identical.

I discovered that physical elements on the weather alter the melatonin/serotonin balance of people, leading to the Foehn Disease and Seasonally Affective Disorder. We showed that the Foehn Disease, a syndrome related to hot, dry winds, melatonin reduction and serotonin enhancement, is related to changes in the local oscillating electric fields. They are created by turbulence in the hot, dry wind with excess positive ions. They change the ground level electric fields and produce symptoms that are totally consistent with reduced melatonin. The enhanced serotonin was identified in urine samples from people in Israel exposed to hot dry desert winds.

In 1994 I was invited to a local primary school where Telecom proposed to install a cell site alongside the infant block. At a school meeting, an industrial and occupational health consultant Dr David Black, assured the parents and teachers that there would be no health effects because the microwave exposure that the children would experience was a small fraction of the standard. This was confirmed by the Technical Manager of Telecom who claimed, with Dr Black, credibility as members of the Standards Committee. Dr Black claims to be independent while he gains over 95% of his income from the telecommunication companies.

I expressed caution, as a biophysicist. I was also an elected Regional Councillor who had to regulate the release of contaminants into the environment. This is under New Zealand's environmental law, the Resource Management Act (RMA), 1991. The Act has a level of evidence threshold of potential effects. It is evidence-based not standards-based. Dr Black said there was no evidence of effects below the standard. I confirmed that I did not know of any evidence at that time. However, I expressed caution because children were more vulnerable than adults, the technology was very new, and if effects were found it would be too late to reverse the problem. That is why the RMA is based on potential effects. Dr Ivan Beale, a Psychologist representing a community group on the Standards Committee, also expressed serious caution.

The meeting voted by 84 % to reject the proposal. They did not respect Dr Black's position and were surprised that Telecom's manager and health consultant were on the Standards committee that was regulating Telecom's activities. The also discovered that the New Zealand, National Radiation Laboratory, was also a consultant to Telecom and BellSouth and the Director, Dr Andrew McEwan, was also one of the 8 people on the New Zealand part of TE/7.

The very next day I went to the Medical School library and carried out a MEDLINE search and found hundreds of studies showing biological, animal, occupational and residential adverse effects. Dr Black was clearly wrong.

Late in 1995 I was asked to form a scientific team to appeal a cell site that was proposed for a residential neighbourhood in Christchurch. In a powerlines case the court had discussed the potential effects evidence level. It stated that it required a plausible biological mechanism [in the absence of actual human studies]. In that case Dr Andrew McEwan (NRL) and Dr Michael Repacholi, appearing for the applicant company, Transpower, assured the court that there were no effects below the standard.

Hence in the cell site appeal case, the McIntyre Case, our scientific evidence was based on Dr Richard Luben, biophysicist and endocrinologist, presenting evidence of biological effects, and Professor John Goldsmith, Epidemiologist, presenting evidence of human health effects. I gave the overview evidence and presented the exposure levels associated with the studies cited. In opposition, appearing for BellSouth, were Mr Martin Gledhill, NRL, and Dr Michael Repacholi, WHO consultant and chairman of ICNIRP. Dr Repacholi's evidence was that there were no adverse health effects unless the exposure raised the temperature by more than 1°C, and the standard protects everyone from that by a factor of 50, i.e. SAR = 0.08 W/kg.

Dr Repacholi was particularly critical of my evidence. For example, I had stated that for mean exposures of less than 1$\mu W/cm^2$, U.S. physiotherapists had experienced significant increases in miscarriage. Dr Repacholi said that this was not plausible because it would mean that if 4 people stood around a pregnant woman she would miscarry. Since this did not happen my evidence could not be true.

Dr Repacholi relied heavily on the WHO 1993 review, that he referred to as "The Consensus of Science". He had chaired the Task group and was the Technical Editor of the Report. The review report refers to a study that concludes that "In the RF range, the black body radiation" is "0.3$\mu W/cm^2$, when integrated up to 300 GHz, Repacholi (1983)."

Dr Repacholi's argument was that 4 x 0.3$\mu W/cm^2$ = 1.2$\mu W/cm^2$, is more than 1$\mu W/cm^2$. Therefore 4 people would cause miscarriage if I was correct. Under cross-examination, he agreed that the microwave part of the spectrum is a very small part of the 0.3 $\mu W/cm^2$. He also agreed that all objects radiated thermal radiation and that the net flow between people would be near zero if they had the similar temperatures. In backing down he described his criticism of my evidence as "only tongue in cheek". This was sworn evidence of a person promoted and presented to the court as a world expert. He swore to tell the "truth, the whole truth and nothing but the truth". I watched as he backed down on claim after claim.

The court's decision rejected Dr Repacholi's assurance that the Standard was protective. The court set the public exposure limit for this case at 2$\mu W/cm^2$. The court also said that this could be revised if there was new evidence. I set about finding the new evidence of effects below 2$\mu W/cm^2$ by researching the published literature, and visiting many universities and laboratories around the world. I found that there was growing evidence of effects well below 2$\mu W/cm^2$, even pointing to zero exposure as the level of no effect.

During this experience I gained significant insights into the way that WHO and ICNIRP were being managed and chaired by Dr Repacholi. The very strongly held views that there were only thermal effects came through consistently and pervasively. I had the confidence of a court decision to back me up that there was evidence of effects below the standard and that the court had rejected Dr Repacholi's position and evidence. I started at an exposure level of 1 % of the standard and worked my way down.

How reliable is ICNIRP and the WHO's Dr Michael Repacholi ?

On these matters I have no respect for the position of ICNIRP nor that of the WHO. The WHO position is taken solely by Dr Repacholi. ICNIRP is a small self-appointed, self-promoted group that claims standing by having WHO recognition. In other words, a body

formed in part and led by Dr Repacholi, claims its standing by being recognized by Dr Repacholi.

I have seen more and more evidence of misrepresentation and deliberate misinformation from ICNIRP and Dr Repacholi. These are strong statements but they are documented. ICNIRP, under Dr Repacholi's chairmanship, prepared several statements during the mid-1990's. They consistently misquote and misrepresent the published research results. They reject all epidemiological evidence because every single epidemiological study occurs with mean exposure levels orders of magnitude below their thermally-based standard.
They are highly selective, using only a small proportion of the available studies in order to construct and defend their own case. They prefer author's conclusions that there are no effects, even when the data and analysis in the paper clash with this and contradict it. They dismiss large, reliable and well defined studies as ill defined and unreliable. They state that studies don't show significant increases in CNS cancers when they actually do, even when the papers include significant dose-response relationships. Both the WHO and ICNIRP, under Dr Repacholi's leadership, have maintained the thermal view to the present, despite the large and ever growing body of scientific research that firmly and conclusively challenges this.

At the recent conference at the European Parliament I noted several claims by Dr Repacholi that are not true. He claimed to be independent of industry and independent of ICNIRP. He said that he didn't go to China with industry representatives to try to persuade the Chinese to relax their standard. He also claimed that WHO conferences produced conclusions from the participants that he had no role in at all. In other forums he has claimed that there is no evidence that cell phones are dangerous and that there are no established non-thermal effects.

Dr Repacholi does have close links with industry. He not only appeared in New Zealand in two court cases for industrial client, in Vienna he was taken to an industry sponsored press conference where he stated that there was no evidence that GSM cellphones were hazardous to health. At the conference he presented his paper on the Telstra funded project that showed that GSM cellphone radiation at quite low non-thermal levels, doubled the cancer in mice. When challenged by the conference chairman, Dr Michael Kundi, Dr Repacholi said that a study is not evidence until it is replicated. The conference rejected this. A study is evidence. Replication provides confirmation and establishment.

After Dr Carl Blackman's paper on calcium ion efflux, which is a well documented phenomena which is well described and extended in over 8 laboratories, Dr Repacholi agreed with all the other scientists at the conference that this was an established non-thermal effect of EMR exposure. However, he wouldn't sign the Vienna Resolution that stated this. He said be couldn't because it was not a WHO organized conference. Later in the year Dr Repacholi convened a WHO workshop on non-thermal effects and it concluded that there were none. Dr John Goldsmith was in the epidemiologic working group. He has published a paper that records the group's deep concern that Dr Repacholi pre-wrote and pre-circulated the conclusions of the workshop. The participants disagreed with the conclusions but Dr Repacholi would not change them. I personally discussed this with Dr Goldsmith and confirmed his experience.

Dr Goldsmith died last year. It is a very sad loss for the public health protection efforts of the world.

Dr Repacholi's trip to China has reported in Microwave News. After his denial in Brussels about going with representatives from industry, I asked Dr Louis Slesin, the highly respected founder and editor of Microwave News about the accuracy of his sources. Dr Slesin replied that he had obtained the information about the trip from the three industrial representatives that went to China with Dr Repacholi.

Dr Repacholi claims to be totally independent of ICNIRP are equally wrong. He assisted with the establishment of ICNIRP and became its second chairman. He is currently Chairman emeritus of ICNIRP.

Our experience of Dr Repacholi in New Zealand, is reinforced time and time again in the international arena. I urge you to be extremely cautious about accepting any of Dr Repacholi's claims.

He claims that I am not credible because I "re-analyse" the data in published papers. He made this claim in front of the Vienna Workshop. I was supported by the workshop because it was accepted that science relies most strongly on data and data analysis. When data and the analysis of data conflict with the published conclusions, then the more classical and appropriate scientific approach requires the use of the data rather than the original conclusions.

A good example of this is given by the U.S. Physiotherapist study. Drs Hocking and Joyner from Telstra wrote a letter to the journal showing that shortwave RF penetrates the uterus and fetus much more strongly than do microwaves, Figure 2.

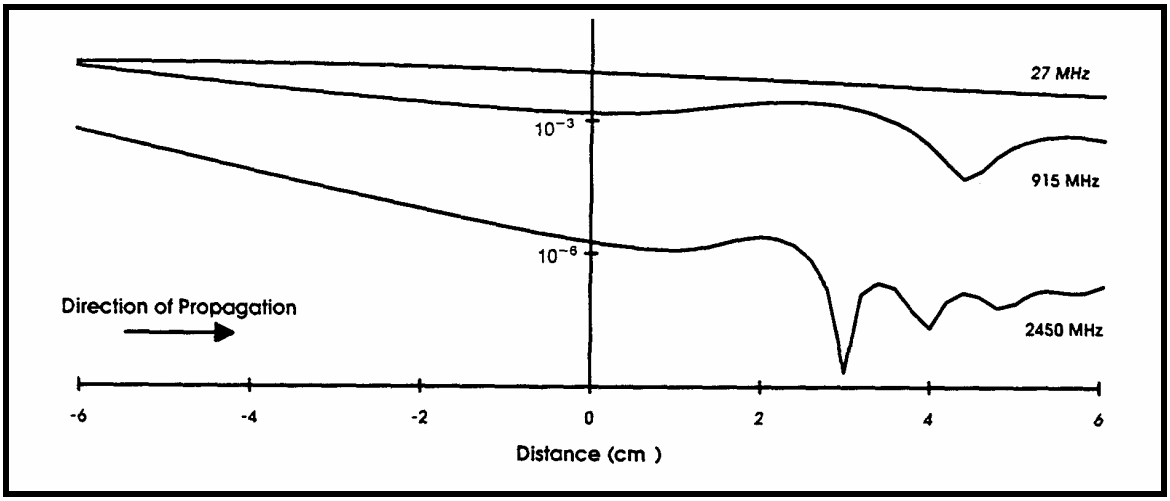

Figure 2: A model developed by Telstra to estimate the penetration and absorption of RF/MW energy in humans. The zero point is the middle of a uterus for a woman exposed to 1 mW/cm^2. The vertical scale is logarithmic.

The model gives the maximum Specific Absorption Rate (SAR) for SW (27.12 MHz) of 0.209 W/kg, for microwave (915 MHz) of 0.023 W/kg and for (2450 MHz) of 0.000027 W/kg.

The study had shown that microwaves significantly increased the incidence of miscarriage in a dose-response manner in the first trimester. Hocking and Joyner used thermal arguments to claimed that the result was wrong because the shortwave exposure of the fetus was far stronger than the microwave exposure.

The original authors, Ouellet-Hellstrom and Stewart (1993) reply **"The data are fixed but the explanations are not!"**

This is clearly not a thermal effect. Sandyk et al. (1992) state that melatonin reduction is a cause of miscarriage.

I used the Hocking and Joyner data to argue with the ARL staff that the miscarriage was not caused by heating but was likely to be due to accumulated chromosome damage. They found this very hard to accept because they strongly hold the thermal view. They finally reluctantly agreed that there was some support for my argument in the Hocking and Joyner data.

The Thermal RF/MW approach is based on the post World War II U.S. Tri-Service program. This had the objective to determine the thermal threshold. It was assumed that the only hazard was burns. In the year 2000 this is not scientifically credible. If there is any available epidemiological studies showing human health effects then they should be the source of human standards. As Professor Abraham Lilienfeld said in 1983:

"The proper study of man is man."

I paraphrase this as:

"Human health standards should be based on human health studies."

My personal experience of the world situation:

It is rare for an individual, such as myself, to be in a position to challenge world authorities. I was very surprised that I was able to show how wrongly they use scientific information and then to be invited into the world forums to present this information. I have done this several times, in stronger and stronger forms. The Europeans are especially concerned to get the best and most reliable information. Italy and Switzerland have recently reduced their standards to 10 and 5$\mu W/cm^2$. The city and province of Salzburg has adopted 0.1$\mu W/cm^2$ for cell sites.

I have spent a great deal of time and our own salary earned money, to try to determine the best and most reliable information about the health effects of EMR. I have collected, analysed, synthesized, integrated and summarized the available biological, animal and human health studies. I have received a great deal of supportive help from the world's leading independent EMR researchers, including Drs John Goldsmith, Richard Luben, Ross Adey, Henry Lai, Carl Blackman, Louis Slesin, Ollie Johansen, Lebrecht von Klitzing, Leif Salford, Michael Kundi, Gerd Oberfeld, and Theo Abelin.

I am now an integral scientific member of the independent international EMR research community. I have participated in over 20 international meetings as a speaker and keynote speaker. Some of these are listed below. This includes the two key Austrian meetings that resulted in the Vienna and Salzburg Resolutions, of which I am a signatory.

You will see that these resolutions are supported by a large body of independent scientists. Together they form a group that is more than three times as big as ICNIRP. They state that there are established biological effects of low level exposure to RF/MW radiation (Vienna), and that cell site exposures should be limited to 0.1μW/cm^2 (Salzburg).

Chronological summary of my international activities in EMR health effects:

June 1996 Poster presentation: "Electromagnetic radiation - Biophysics, Epidemiology, Health effects and Law - A New Zealand perspective.

1996/97 Study-leave international research tour (22 visits).

Feb/Mar 97 Australian Lecture tour, Sydney, Canberra, Melbourne and Adelaide. Evidence of health effects from EMR.

Mar 1998 United States Lecture tour, Boston, Golden Colorado, and San Francisco, Actual and potential effects of electromagnetic radiation below 2μW/cm^2.

Oct 1998 "Should cell phones have health warnings - the risk of brain tumours". Scientific Workshop and the Health Effects of EMF, University of Vienna, Vienna, Austria. Signatory to the Vienna Resolution.

Feb 1999 "Criticism of the proposal to adopt the ICNIRP Guidelines for cell sites in New Zealand". A peer-review report to the Ministries of Health and Environment.

Apr 1999 "A new paradigm: the biological effects of EMR", Community Conference at Tiburon, California.

Aug 1999 "EMR Effects on Breast Cancer". 2nd World Breast Cancer Conference, Ottawa, Canada.

Nov 1999 "Setting EMR standards based on Epidemiology - ICNIRP Critique". Italian National Congress on EMR Health Effects, Trento, Italy. Followed by 2 presentations in Rome, and one each in Avellino, Perugia and Florence.

May 2000 Evidence that electromagnetic radiation is genotoxic: the implications for the epidemiology of cancer and cardiac, neurological and reproductive effects". Beehive Theatrette, NZ Parliament.

June 2000 "Living near broadcast towers is hazardous to your health: Implications for cell sites." CODACOMS (Italian national consumer organisation) National Congress on EMR health effects, Rocarasso, Italy.

June 2000 "Probable health effects associated with mobile phone base stations in communities: the need for health studies". Salzburg Cell site EMR Conference, Salzburg, Austria. Signatory to the Salzburg Resolution.

June 2000 Evidence that electromagnetic radiation is genotoxic: the implications for the epidemiology of cancer and cardiac, neurological and reproductive effects". Public lecture and national media interviews, Dublin, Ireland.

June 2000 Evidence that electromagnetic radiation is genotoxic: the implications for the epidemiology of cancer and cardiac, neurological and reproductive effects". European Parliament Conference, Brussels.

It is now my pleasure to present to you the latest information that I have presented around the world, showing that we are closely tuned into the natural environment and this can be interfered with by natural and artificial EMR down to levels that are very close to zero exposure. I will also demonstrate the constructive dismissal approach of ICNIRP and WHO as they try to defend their thermal view at all costs.

Humans are tuned into the Natural EMR:

In a very real and scientifically sound approach we can describe the internal communications systems of the human body in a parallel with a nationwide telecommunications system with local stations and local receivers. The brain in the central telecommunications centre. The heart and other muscles and organs are local centres, and cells are individual receivers. The central nervous system provides a "cable" system to carry electrical signals to body organs. The brain also uses first messengers that flow through the blood and other systems to communicate with cells.

Cells have aerials (receptors), that receive the signal and amplify it by 100,000 to 1 million times, and then it communicates with the cell a particular message. Cells communicate with cells through external signals such as ions, and ion currents carrying FM encoded signals through the gap junctions that help muscles and other solid tissue to communicate and remain in harmony with each other. As is necessary for each heart beat or any muscle movement. Cells have ion oscillators that encode and decode AM, FM and digital signals, just like individual radios and TVs. These oscillators, encoders and decoders use ELF frequencies < 30 Hz.

The brain is like a central computer, TV image processing centre and receiving centre. It receives external signals through the sensors, sights, sounds, smells, and feelings. The sensors send a stream of messages to the brain where they are converted to bioelectromagnetic signals, EEG, and processed and memorized. Reactions of happiness, sadness, excitement, embarrassment, etc are communicated to our heart, muscles and cells through the internal telecommunications system.

The brain is also an FM radio receiver. The parallel is a shortwave radio receiver that is picking up the BBC on the other side of the world. This is happening even though the local EMR environment has a strong static geomagnetic field and powerful radio and TV stations and cell sites from local transmitters, together producing a local RF/MW field over a billion times higher than the shortwave signal. This remote SW signal can be tuned into using an oscillator that is tuned to the carrier frequency of the SW signal. Then resonant absorption occurs, the signal is received, decoded, amplified and heard as BBC World News, My Word or music.

EEG-Schumann Resonance Frequency Matching:

Our brain's EEG shares the same frequency range with the Schumann Resonance Spectrum, Figure 3.

A basic feature of a receiver and its decoder is the ability to detect and absorb the incoming signal through frequency matching, tuned oscillators and decoding circuitry. Figure 3 shows a typical human EEG spectrum.

The frequency ranges of the EEG and the Schumann Resonance Spectrum exactly coincide. This provides physical plausibility for resonant absorption. There is even a suggestion of direct coincidence of the four higher frequency peaks, especially if they were slightly higher. When it is noted that diurnal changes in the typical EEG pattern also follow diurnal changes in the Schumann Resonance Spectrum, it begins to look more like "design" than coincidence. We have neurological frequency matching. Is there a tuning ability?

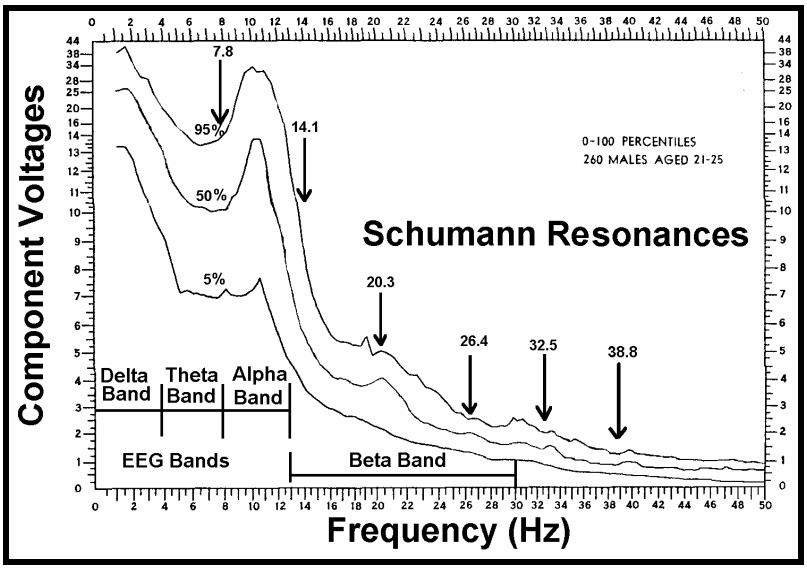

Figure 3: Comparison of the frequency spectra of the human EEG from 260 young males showing the 5%, 50% and 95%ile bands, adapted from Gibbs and Gibbs (1951), and Schumann Resonance peaks, from Polk (1982).

Brains have tuned circuits involving calcium ions and hormones. They also have bioelectrochemical phase-lock loop circuits to detect and respond to incoming ELF signals, Ahissar, Haidarliu and Zackerhouse (1997), just like FM radios, Motluk (1997). Such feedback loops were also identified in the dorsal giant interneurons of an insect, Libersat, Levy and Camhi (1989).

Hence human and animal brains can detect and tune into incoming ELF signals.

German research in the 1950's and 1960's proved that we do tune into these signals, detect them and react to them through reaction time change. We use them to synchronize our circadian rhythm, Konig (1974), Figure 4, and Wever (1974).

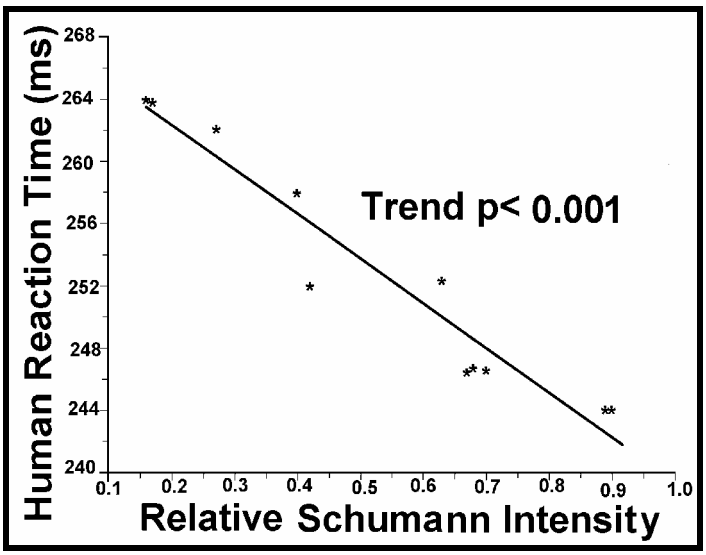

Figure 4: Human reaction times as a function of Schumann Resonance 8-10 Hz Relative Intensity, for 49,500 subjects tested during 18 days in September 1953, at the German Traffic exhibition in Munich. Derived from data in Figure 3 of König (1974b). Trend: t = 10.414, 2-tailed p<0.001.

Konig (1974) found that human reaction times were extremely significantly related to the intensity of the Schumann Resonances and local sferics signals (from local thunderstorms). This was confirmed in laboratory experiments with human volunteers and independently confirmed in the U.S., Hamer (1965, 1969). They found that extremely small signals of 10 Hz speeds up the reaction time and 3 Hz slows it down. The signal used by Hamer in his laboratory was almost as small as the Schumann Intensity of 1 mV/m. This converts to 0.3pW/cm^2. A picoWatt (pW) is one millionth of a microWatt (μW).

When there is a solar storm, a Geomagnetic Event is experienced on earth, with enhanced Geomagnetic Indices, enhanced ionospheric ion concentrations and enhanced Schumann Resonance Intensity, Figure 5. This shows the Schumann Resonance signal, measured at UC Berkeley, averaged over 24 GMA events during 1997.

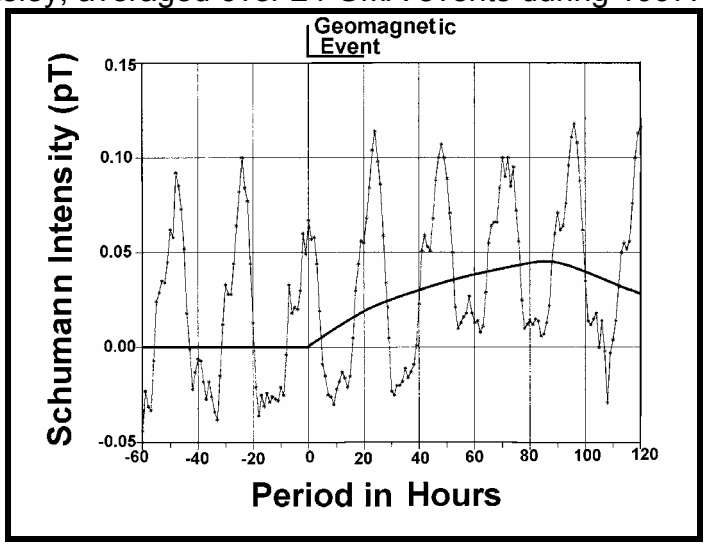

Figure 5: Superimposed epoch analysis of the impact of a Geomagnetic events, as defined by the Ap Index, for the anomaly in the Schumann Resonance 0-20 Hz intensity for 24 events in 1997. The solid line is a smoothed daily mean curve.

The higher intensity, especially at night, will change the human reactions. One reaction could be seen by a reduction in melatonin. Burch et al. (1999) found an extremely significant dose-response reduction in human melatonin in association with GMA indices, Figure 6.

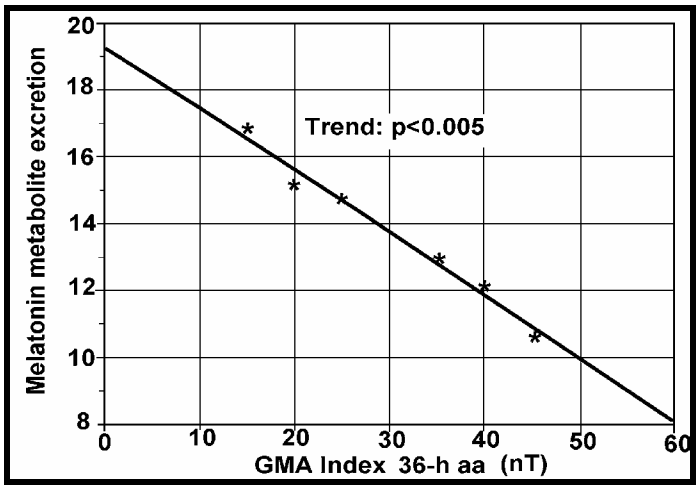

Figure 6: Reduction in the melatonin metabolite 6-OHMS in urine from electric utility workers, as a function of the 36 hr global GMA aa-index, Burch et al. (1999).

This is a powerful set of strongly internally consistent, extremely significant, observations that show that human brains can and do detect and react to Schumann Resonance signals. When the signal increases then melatonin decreases. Therefore geomagnetic activity (GMA) is genotoxic and can produce all of the adverse health effects associated with melatonin reduction. This is confirmed by a large body of Studies summarised in my Schumann Resonance paper that is in Press in the Journal of Natural Hazards, Cherry (2000). I have shown that GMA is a natural hazard for human health. The plausible mechanism involves the Schumann Resonance signal that has a field strength of 1 mV/m and an intensity of 0.3 pW/cm^2 on average.

The data shows, in many studies, that vulnerable people get ill and die when GMA raises and lowers the Schumann Intensity. Hence there is an optimal level and too little and too much causes problems. The same is true of melatonin, temperature, food, minerals, etc.. It is a biological concept called Homeostasis. The natural electromagnetic environment helps to maintain the Homeostasis of the internal electromagnetic environment. This includes synchronization of the ELF EEG signals in the brain and ECG signals in the heart. GMA is associated with neurological and cardiac illness and death.

One of the most important single studies involved cot death (Sudden Infant Death Syndrome) in Ontario, Canada. O'Connor and Persinger (1997) were investigating the GMA melatonin hypothesis by seeing if a melatonin-related syndrome (SIDS) varied with GMA. They found that SIDS incidence significantly increased when GMA >30 nT and GMA < 20 nT, - a homeostatic result. This confirms that GMA causes illness and death in vulnerable people, babies, and involves melatonin homeostasis.

This shows that very young babies are sensitive to variations in the natural EMR at extremely low exposure levels. Thus we would expect the fetus to also be vulnerable.

Ten epidemiological studies have found significant miscarriage from EMR exposure across the spectrum from ELF, SW, to RF/MW. The Scandanavian physiotherapist studies, Kallen et al. (1982) and Larsen et al. (1991) also found significant prematurity,

congenital malformation, still birth and cot death. Ouellet-Hellstrom and Stewart (1993) confirm the causal relationship with a highly significant dose-response relationship.

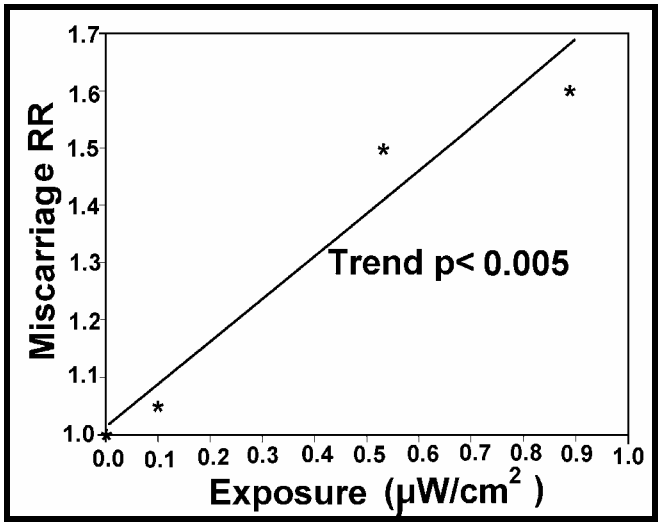

Figure 8: Microwave exposure associated first trimester miscarriage, Ouellet-Hellstrom and Stewart (1993). Exposure is monthly mean exposure based on treatments/month, 3 minutes/treatment at 600 $\mu W/cm^2$.

Figure 8 shows, consistent with EMR being genotoxic and damaging the chromosomes cell by cell, that the threshold of effect is zero exposure. Note that one of the microwave frequencies, 915 MHz, is in the cellphone range. The difference is that the diathermy used constant signals while cellphones use modulated and pulsed signals that are generally much more damaging.

A Greek study confirms the evidence that chronic low level exposure to RF radiation leads to reproductive problems. Magras and Xenos (1997) responded to health concerns among residents living in the vicinity of an RF transmission tower in Greece, by placing groups of mice at various locations in relation to the tower. The mice fertility was monitored over several generations and related to the RF exposure. Figure 9 shows the fertility rate of the two exposed groups.

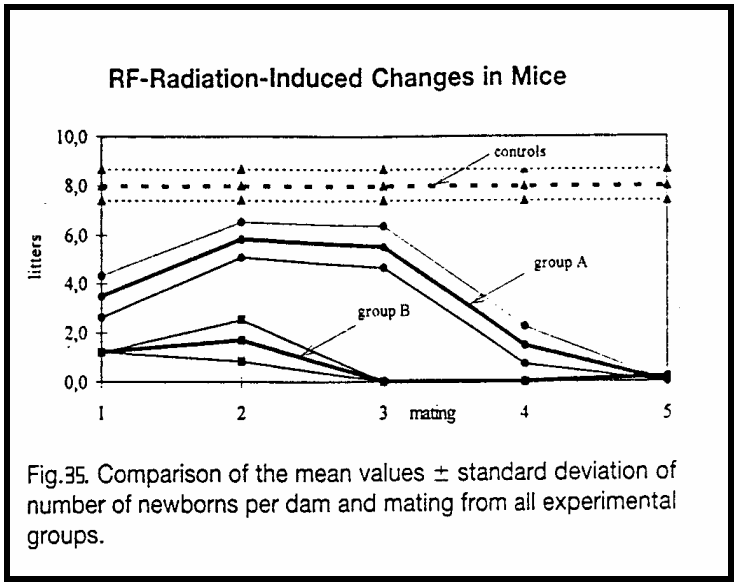

Fig.35. Comparison of the mean values ± standard deviation of number of newborns per dam and mating from all experimental groups.

Figure 9: Multigenerational exposure of mice to low level RF leads to complete infertility, Magras and Xenos (1997).

Where group A the "Low" exposure group, 0.168μW/cm^2, became infertile after 5 generations and B the "High" exposure group, 1.053μW/cm^2, became infertile after only 3 generations. This is a highly significant result because so few multi-generation studies have been done and the effects of this study occur at extremely low levels and the effect is total infertility.

EMR Spectrum Principle:

It is also important to note that if an effect is seen with low frequency signals, such as an ELF 50 Hz or 60 Hz signal, or the Schumann Resonance ELF signals, then it is more likely and likely to be worse for modulated or pulsed RF/MW. This is because an ELF signal has a very long wavelength and generally passes easily right through the body. Unless there is a resonant oscillator, such as for the Schumann Resonances, it induces quite small fields in the body. On the other hand the RF/MW signals have wavelengths closer to the dimensions of bodies and body parts, they are more strongly absorbed in human bodies through the aerial effect. They also couple more strongly into the tissues as shown by the nearly linearly decreasing dielectric constant. This is like the resistance for oscillating signals. The following figure shows the induced current for a unit imposed field, as the carrier frequency increases.

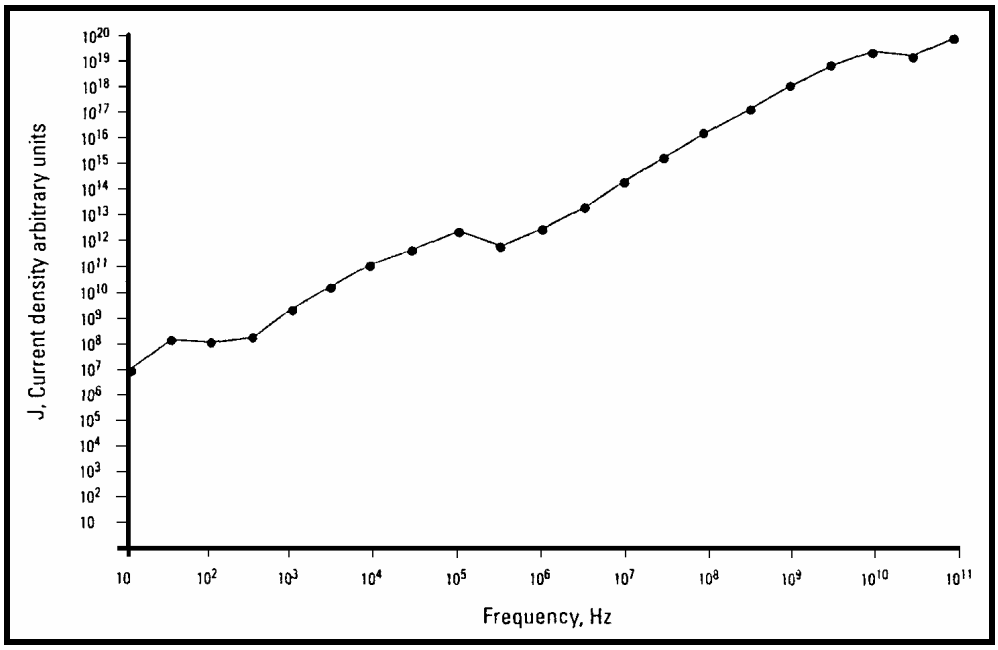

Figure 10: Induced tissue current from a unit applied field, as a function of the carrier frequency, Vignati and Giuliani (1997).

I call this the EMR Spectrum Principle. It was confirmed by Adey (1989). He showed that a 57 V/m ELF signal induced a tissue and electric field of 10^{-7} V/cm in a chick brain, and a 57 V/m 147 MHz signal with ELF modulation induced a field of 10^{-1} V/cm, 1 million times higher.

Public Health Assessment Approach:

Bradford Hill Approach:

The assessment of evidence approach used to evaluating the cause-and-effect association between Schumann Resonances, human reactions and health effects, is the classic assessment outlined by Sir Austin Bradford Hill, Hill (1965).

Sir Austin outlines and describes nine "view points" to assist with this assessment. The effect must take place after the exposure. Strength of Association and Dose-Response Relationship give strong evidence but their absence is not a limitation. Consistency, Analogy and Experiment can contribute.

One of Sir Austin's conclusions is that even a consistent set of observed non-significant associations can, under some circumstances, be assessed as causal. He makes it clear that in human studies of health effects, scientific proof is not the approach to apply. Rather, it is an assessment of evidence approach. He summarizes the approach as:

"Here then are nine different viewpoints from all of which we should study association before we cry causation. What I do not believe - and this has been suggested - is that we can usefully lay down some hard-and-fast rules of evidence that *must* be obeyed before we accept cause-and-effect. None of my nine viewpoints can bring indisputable evidence for or against the cause-and-effect hypothesis and none can be required as a *sine qua non.* What they can do, with greater or less strength, is to help us to make up our minds on the fundamental question - is there any other way of explaining the set of facts before us, is there any other answer equally, or more likely than cause-and-effect?", Hill (1965).

The dose-response relationship gives the strongest conclusion of cause-and effect. Sir Austin says that "the clear dose-response curve admits of a simple explanation and obviously puts the case in a clearer light." That is, it is indicative of cause-and -effect.

The ELF Public Health Context:

Dose-response relationships for childhood leukaemia associated with living in the vicinity of high voltage powerlines identifies a threshold of about 0.1μT, Feychting et al. (1995).

S-GMA activity is primarily associated with neurological and cardiac effects. This is reasonable because the brain and heart are electromagnetically controlled.

A recent study, Savitz et al. (1999), shows that electric utility workers show dose-response increases in heart attack and arrhythmic heart disease mortality, based on cumulative μT-yrs of occupational exposure. These have thresholds near zero exposure.

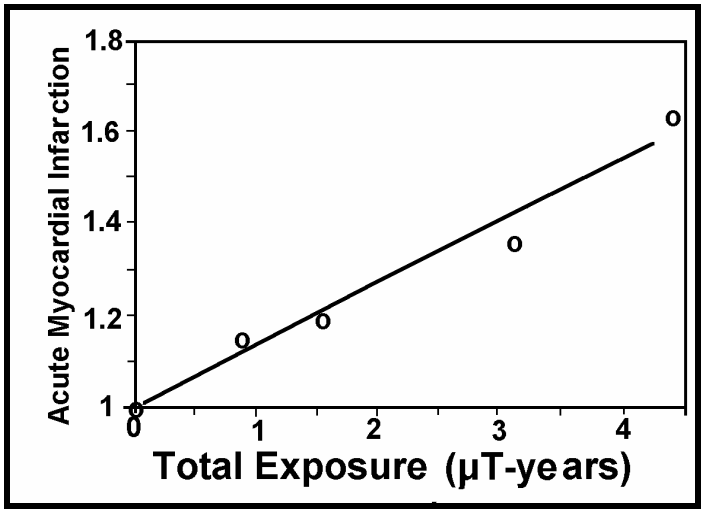

Figure 11: Heart Attack (acute myocardial infarction) as a function of mean exposure for electric utility workers in the U.S., Savitz et al. (1999).

A second study investigated the incidence of suicide in electric utility workers, Van Wijngaarden et al. (2000). Over 2800 people were personally monitored for magnetic field exposure. This showed an exposure-based significant dose-response increase in suicide in the current year and in the past 1-5 years. The lowest exposure group, with a 19% increase in suicide, were assessed to have annual mean exposures in the range 0-0.029μT. There is a non-linear dose-response relationship that has a threshold at zero exposure.

These show convincingly that the safe ELF exposure level is zero, whereas the ICNIRP guideline for the public is 100μT. The ELF guideline is based on avoiding electric shock form acute exposure. It does not protect from highly significant health effects such as cancer, heart attack or suicide from chronic, day-by -day and month-by-month exposures that damage cells in the body, heart and brain, interfering with the natural control signals and causing illness and death with a threshold of zero exposure. A chronic exposure to 0.1μT over a year is associated with a 60 % increase in suicide.

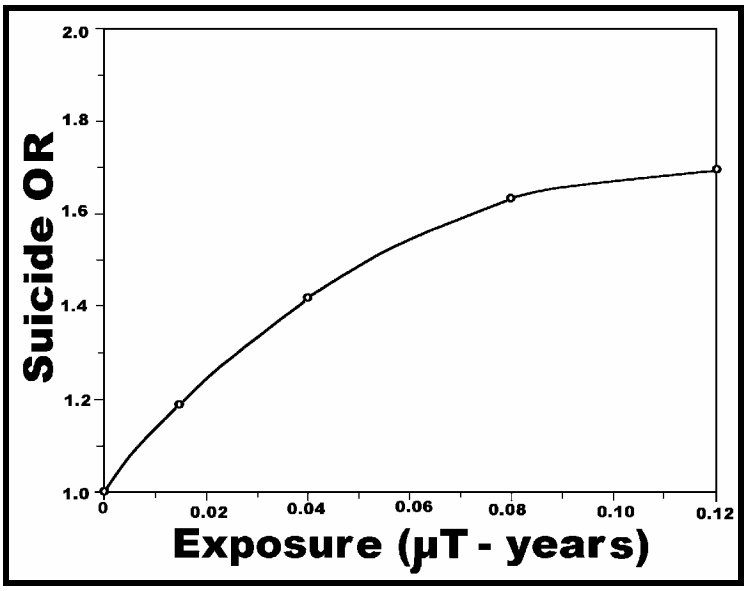

Figure 12: Suicide in electric utility workers in the United States as a function of their mean ELF exposure over the year prior to their suicide, van Wijngaarden et al. (1999).

Suicide is a result of deep clinical depression. High Voltage powerline exposure is associated with clinical depression, Verkasalo et al. (1997), and suicide, Perry et al. (1981). Baris and Armstrong (1990) found a highly significant increase in suicide among Telegraph radio operators in England and Wales. This is a melatonin reduction-related effect and the threshold is consistent with the studies cited here about S-GMA related effects with the Schumann Resonance signal involved as the proposed biophysical mechanism.

These recently published studies identify dose-responses for arrhythmia related heart disease problem and a melatonin reduction related neurological effect. This gives strong support for a biological mechanism that relates to timing synchronization and melatonin reduction. They also give evidence supporting a threshold of effect near zero ELF intensity exposure.

Evidence that EMR is Genotoxic:

Many studies have shown that radiofrequency/microwave (RF/MW) radiation and extremely low frequency (ELF) fields cause increased DNA strand breakage and chromosome aberrations. This has been shown in cell lines, human blood, animals and living human beings. This means that epidemiological studies of people exposed to electromagnetic radiation (EMR) are likely to show increased cancer, miscarriage and reproductive adverse effects. In fact many epidemiological studies have shown these effects, Goldsmith (1995, 1996, 1997, 1997a), Szmigielski (1991, 1996).

Two plausible biological mechanisms involving free radicals are involved in this effect. The first involves increased free radical activity and genetic damage as a response to exposure. The second involves increased free radical activity and genetic damage because of an induced reduction of a free radical scavenger, e.g. reduced melatonin, Reiter (1994). It is clear however, that both mechanisms have the same effect of damaging the DNA and chromosomes. Another established biological mechanism, EMR-induced alteration of cellular calcium ion homeostasis, Blackman (1990), is also involved in cell regulation, cell survival and apoptosis, DNA synthesis and melatonin regulation.

Substances that damage cellular genetic material, such as DNA and chromosomes, are called “genotoxic”. Genotoxic substances cause cancer, reproductive health effects and neurological damage. Chromosome aberrations are visible through powerful microscopes. Chromosomes are formed from folded segments of DNA. Damage to chromosomes is therefore evidence of damage to DNA.

DNA is frequently damaged by natural processes, such as oxygen free radicals. Gey (1993) comments that free radicals may be involved in the etiology of cancer and cardiovascular diseases. In epidemiological studies poor plasma levels of antioxidants (free radical scavengers) are associated with increased relative risks of cancer and ischemic heart disease. Cells have elaborate DNA repair mechanisms because DNA stability is vital for species survival. Uncorrected DNA damage is mutation, Alberts et al. (1994). Alberts et al. outline many DNA repair mechanisms, including Repair Enzymes. They also outline the way apoptosis can digest and destroy damaged cells by internal

"programming" of the process. The Immune System has B lymphocytes that produce antibody proteins to protect against 'foreign' cells, such as mutated cells. Natural Killer (NK) cells kill some types of tumours and some virus-infected cells, Alberts et al.

Enhanced DNA strand breakage leads to enhanced DNA repair. Hence enhanced DNA repair rates are also used as evidence of DNA damage, Meltz (1995).

Direct measurements of Chromosome aberrations:

Direct evidence that EMR induces significant increases in chromosome damage, with significant dose response relationships, is evidence of a causal effect when replicated or extended by independent laboratories.

Chromosome damage from RF/MW exposure:

The first identified study that showed that pulsed RF radiation cause significant chromosome aberrations was Heller and Teixeira-Pinto (1959). Garlic roots were exposed to 27 MHz pulsed at 80 to 180 Hz. for 5 min. They were examined 24 hrs later. They concluded that this RF signal mimicked the chromosomal aberration produced by ionizing radiation and c-mitotic substances. No increased temperature was observed.

Blood samples were taken from the staff of the U.S. Embassy in Moscow. They had been chronically exposed to a low intensity radar signal. Significant increases in chromosome damage was reported, Tonascia and Tonascia (1966) cited in Goldsmith (1997a).

Garaj-Vrhovac et al. (1990) noted the differences and similarities between the mutagenicity of microwaves and VCM (vinyl chloride monomer). They studied a group of workers who were exposed to 10 to 50 $\mu W/cm^2$ of radar produced microwaves. Some were also exposed to about 5 ppm of VCM, a known carcinogen. Exposure to each of these substances (microwaves and VCM) produced highly significant ($p<0.01$ to $p<0.001$) increases in Chromatid breaks, Chromosome breaks, acentric and dicentric breaks in human lymphocytes from blood taken from exposed workers. The results were consistent across two assays, a micronucleus test and chromosome aberration assay. Chromosome aberrations and micronuclei are significantly higher than the controls, ($p<0.05$, $p<0.001$, $p<0.0001$), for each of the exposure intensity.

Garaj-Vrhovac, Horvat and Koren (1991) exposed Chinese hamster cells to 7.7 GHz microwave radiation to determine cell survival and chromosome damage. They assayed chromosome aberrations and micronuclei and found that microwaves increased these in a dose response manner, Figure 13, to levels that were highly significantly elevated ($p<0.02$ to $p<0.01$).

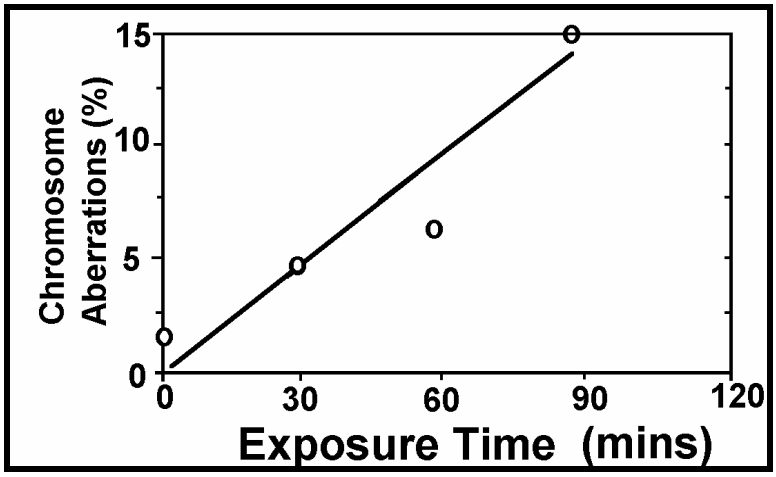

Figure 13: Chromosome aberrations in V79 Chinese hamster cells exposed to 7.7 GHz microwaves at 30 mW/cm^2, Garaj-Vrhovac, Horvat and Koren (1991).

An exposure level of 30 mW/cm^2 is usually able to slightly raise the temperature over an hour. This experiment was undertaken under isothermal conditions, with samples being kept within 0.4°C of 22°C. The consistency of the time exposure and the survival assay at non-thermal exposure levels, confirms that this is a non-thermal effect.

This is very strong evidence of genotoxic effects from RF/MW exposures. When chromosomes are damaged one of the primary protective measures is for the immune system natural killer cells to eliminate the damaged cells. Alternatively the cells can enter programmed cell suicide, apoptosis. Garaj-Vrhovac, Horvat and Koren (1991) measured the cell survival rates. They found that cell survival reduced and the cell death increased in a time dependent and exposure dose response manner, Figure 14.

Figure 14 shows that cell death varies with time and intensity of exposure, down to very low exposure levels. An apparent 'saturation' at high levels also becoming evident. This is probably because of the lethal effect of high intensity microwaves. Since this is an isothermal experiment it raised important questions about the reasons for the cell death as acute genetic damage which is continuously related to microwave exposure down to non-thermal levels.

Note that the general public ICNIRP guideline for microwaves above 2 GHz is 1 mW/cm^2, and for workers is 5 mW/cm^2. Even at 100 times below the public exposure guideline a 60 minute exposure kills 28% of the cells and 30 minutes kills 8 % of the cells. Garaj-Vrhovac et al. (1992) exposed human lymphocytes and showed that microwave radiation produced a dose response increase in chromosome aberrations, Figure 15.

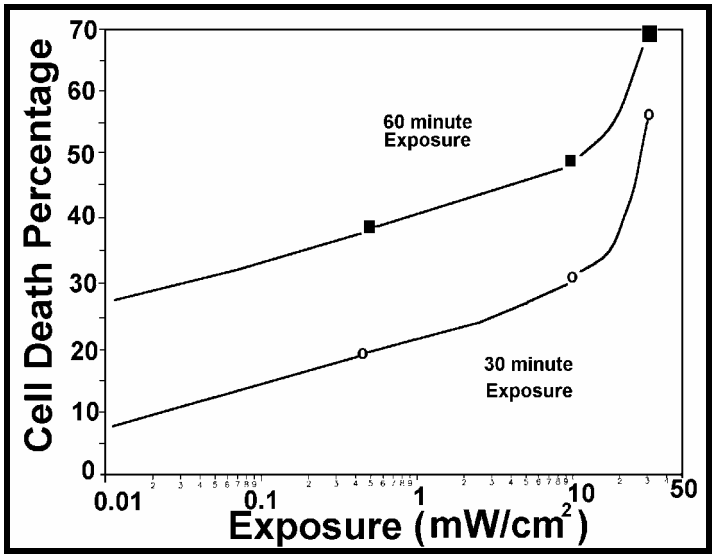

Figure 14: Cell death percentage of Chinese hamster cells exposed to 7.7 GHz microwaves (CW) for 30 minutes and 60 minutes in an isothermal exposure system, Garaj-Vrhovac, Horvat and Koren (1991).

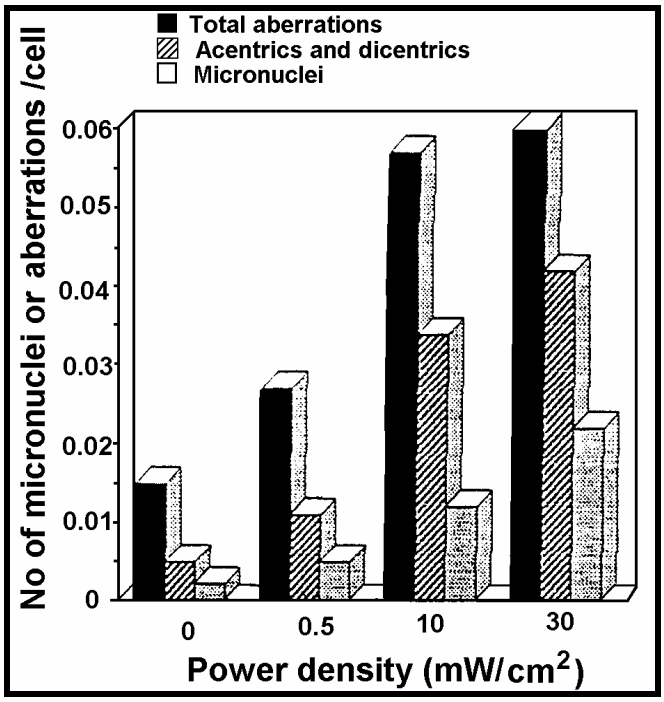

Figure 15: The relation of total chromosome aberrations. micronuclei and specific chromosome aberrations for each cell in human lymphocyte cultures in the dose of microwave radiation in vitro, Garaj-Vrhovac et al. (1992).

Having established that microwave exposure damaged chromosomes, this research group were asked to analyze blood samples from workers who had been exposed to pulsed microwaves generated by air traffic control radars while they were repairing them. Garag-Vrhovac and Fucic (1993) analysed the chromosome aberration (CA) in 6 technical staff who had experienced accidental exposure to the radar. The initial CA percentage ranged from 3% to 33%, all being significantly higher than unexposed people. The repair rate over time was monitored. Figure 16 shows the man who had 33 % CA which was followed over 30 weeks following this exposure. The repair rate follows a significant linear rate (r=0.98), dropping from 33% to 3% over 30 weeks, i.e. 1 %/week.

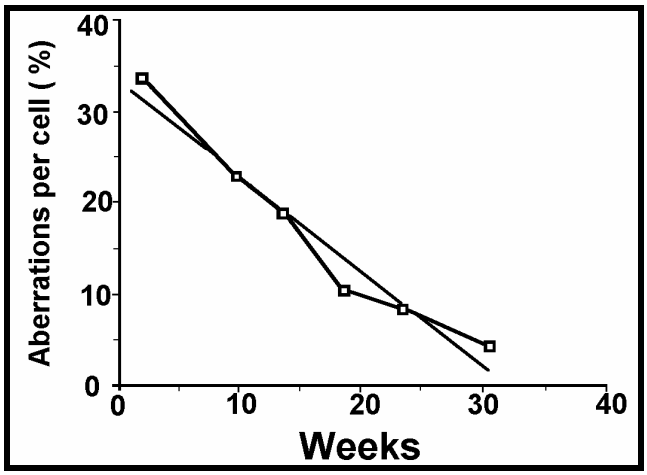

Figure 16: The time-dependent decrease in the number of chromosome aberrations for subjects with high numbers of chromosomal impairments, y = 0.318 - 0.010x, r=0.98. Garaj-Vrhovac and Fucic (1993).

Garaj-Vrhovac (1999) found that 12 workers occupationally exposed to microwave had significantly increased chromosome damage as well as disturbances in the distribution of cells over the first, second and third mitotic divisions.

Quite independently, Maes et al. (1993) found highly significant ($p<0.001$) increases is the frequency of chromosome aberrations (including dicentric and acentric fragments) and micronuclei in human blood exposed to 2.45 GHz microwaves to 30 to 120 minutes in vitro. The micronuclei assay showed a dose response with time, Figure 17.

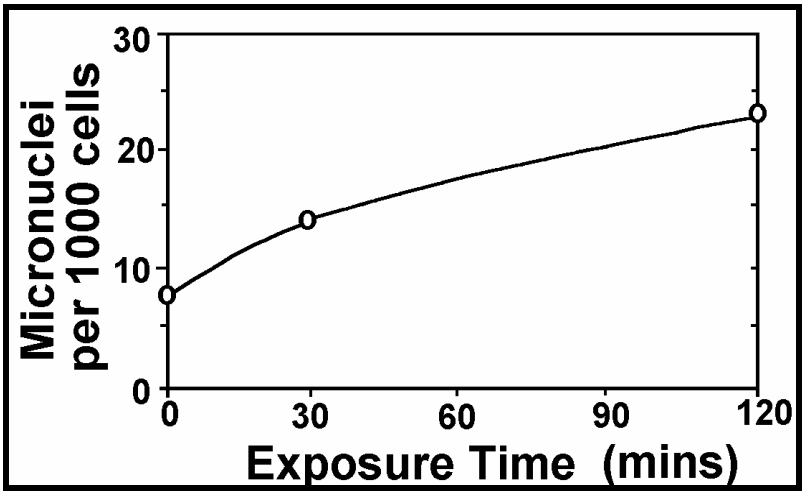

Figure 17: Micronuclei in microwave exposed human lymphocytes, the average of 4 donors, Maes et al. (1993). Exposure was to 75 W/kg, 2.45 GHz microwaves pulsed at 50 Hz, under controlled isothermal conditions

Timchenko and Ianchevskaia (1995), Balode (1996), Haider et al. (1994) and Vijayalaxmi et al. (1997) have reported significant chromosome aberrations from RF/MW exposures. In the Mar/Apr 1999 edition of Microwave News it is reported that Drs Tice, Hook and McRee showed chromosome damage from all cell phones tested, all being statistically significant and all but one highly significant with dose-response relationships up to a factor of three increase in chromosome aberrations.

Vijayalaxmi et al. (1997) chronically exposed cancer prone mice to 2.45 GHz CW microwaves at an SAR of 1 W/kg for 20 hr/day, 7 days/week for 18 months. Their aim was to determine whether microwaves were genotoxic through determining if there was significant chromosome damage. They found highly significant increases in micronuclei in peripheral blood, from 8 per 2000 cells in sham exposed mice to 9 per 2000 cells microwave exposed mice, and increase of 12.5 %, $p<0.001$. There was a significant increase of 6.6%, $p<0.025$, of micronuclei in the bone marrow. They also observed a significant 41 % increase in tumours in the exposed mice compared to the sham exposed mice.

This was a totally unexpected result from this group. A great deal of effort was put into playing down the implications. They describe the increase in peripheral blood as a 0.05%, by dividing the increase of 1 by 2000. This is not a significant increase and this is not the right comparison. It is a deliberate attempt to disguise their true result that shows that microwaves are genotoxic.

Multiple independent studies, in 15 papers, show significant increases in chromosome aberrations from RF/MW exposure. Four studies show dose-response relationships. This is more than adequate to classify RF/MW radiation as genotoxic.

Chromosome damage from ELF exposure:

El Nahas and Oraby (1989) observed significant dose-response dependent micronuclei increase in 50 Hz exposed mice somatic cells. Elevated CA have been recorded in a number of workers in electrical occupations. In Sweden Nordenson et al. (1988) found significant CA in 400 kV-substation workers and with 50 Hz exposures to peripheral human lymphocytes, Nordenson et al. (1984) and human amniotic cells, Nordenson et al. (1994). Significant CA in human lymphocytes exposed to 50 Hz fields are also reported by Rosenthal and Obe (1989), Khalil and Qassem (1991), Garcia-Sagredo and Monteagudo (1991), Valjus et al. (1993) and Skyberg et al. (1993). Skyberg et al. collected their samples from high-voltage laboratory cable splicers and Valjus et al. from power linesmen.

Hence chromosome damage has been recorded from exposes across the EMR spectrum from ELF to RF/MW exposures, in plants, mammal and human cells, animals and human beings, and from many independent laboratories. This confirms that EMR does damage chromosomes and establishes EMR induced chromosome aberrations as a biological effect.

Chromosome Aberrations Conclusions:

Many studies, from independent laboratories, have shown that ELF, RF/MW and cell phone radiation, significantly increases chromosome aberrations in exposed cells, including cells taken from human beings who have been exposed to EMR in occupational situations. Even at very low intensity radar exposures that were experienced at the U.S. Embassy in Moscow, significant increases in chromosome damage was measured from human blood samples. This evidence shows conclusively that across the EMR spectrum, EMR is genotoxic. Hence it is carcinogenic and teratogenic.

Direct evidence of neoplasm in microwave exposed cells:

For a neoplastic cell to survive it must have an altered genetic structure to store the damage and to hide this from the immune system so that NK cells do not kill the neoplasm transformed cells. Balcer-Kubiczek and Harrison (1991) observed a significant dose response increase of neoplastic transformation in a standard cell set (C3H/10T1/2) from a 24 hr exposure to 2.45 GHz microwaves. The transformation was assayed after 8 weeks of exposure to a known cancer promoter chemical TPA, Figure 18. The method was confirmed with a positive control using X-rays. This also showed that 60Hz magnetic fields also significantly increased neoplastic transformation.

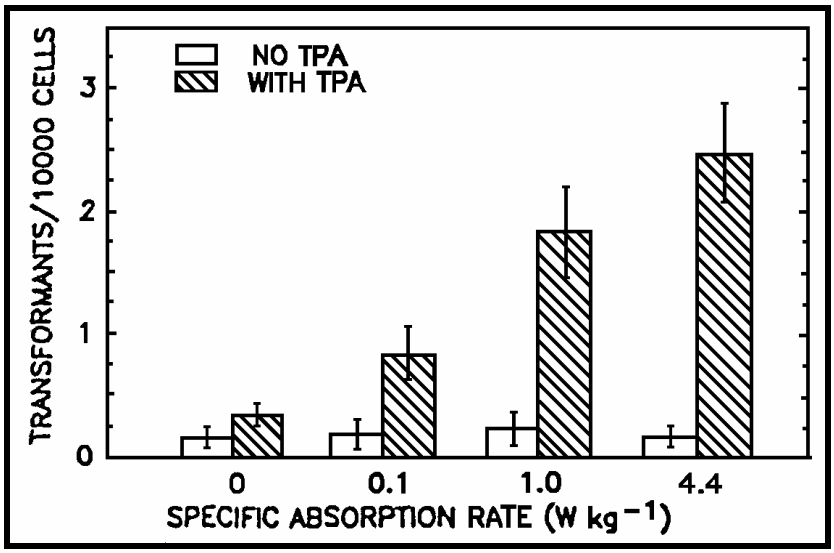

Figure 18: Dose-response relationship for induction of neoplastic transformation in C3H/10T1/2 cells by a 24h exposure to 2.45 GHz microwaves at the specific absorption rate (SAR) with and without TPA post-treatment for 8 weeks, Balcer-Kubiczek and Harrison (1991).

Direct evidence of DNA strand breakage:

Sarkar, Ali and Behari (1994) investigated the effect on DNA of exposures accepted a safe by the Non-ionizing Radiation Committee of IRPA (International Radiation Protection Association - the predecessor of ICNIRP).

The exposure regime was a 2 hr exposure to 2.45 GHz CW microwaves at 1 mW/cm^2, SAR = 1.18 W/kg. They observed significant alterations in the DNA from rat brains and testis in the 7 to 8 kb region of the DNA in the hybridization profile and in a densitometric analysis, Figure 19.

The Comet Assay Method:

A very advanced assay of DNA strand breakage has been developed by Dr N.P. Singh at the University of Washington. This is called the microgel electrophoresis or Comet Assay, Singh et al. (1994). The Comet Assay involves migration of segments of DNA down an electric field gradient, Figure 20.

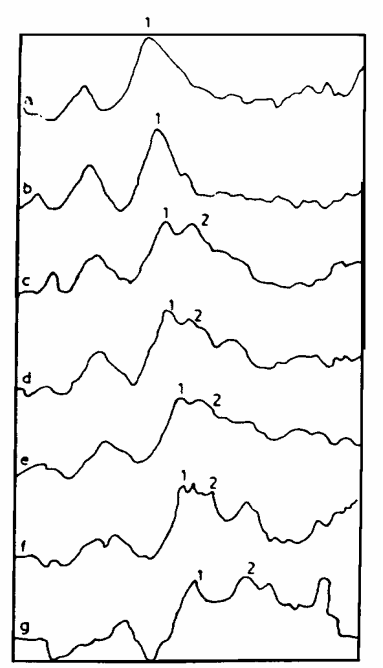

Figure 19: Densitometric analysis of the brain DNA, a and b are control DNA, c to g are DNA from exposed animals. Peak 1 is present in both control and exposed animals while peak 2 appears only in all of the exposed animals.

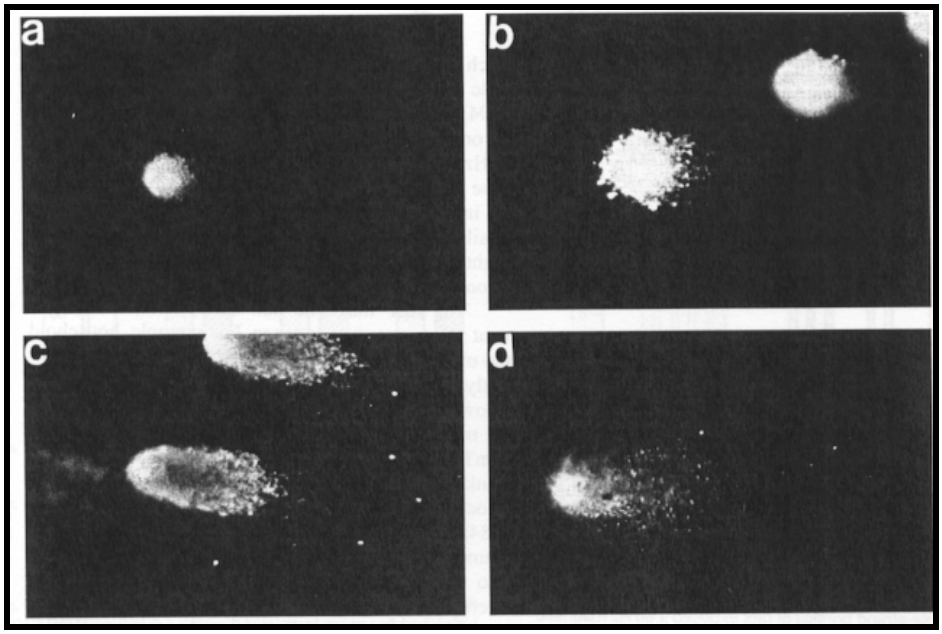

Figure 20: Photographs of double-strand break DNA migration pattern of individual brain cells from rats exposed to (a) bucking condition (0.1 mT), (b) magnetic fields of 0.1 mT, (c) 0.25 mT and (d) 0.5 mT, Lai and Singh (1997a). The “bucking mode” is the condition to reverse the field to cancel the magnetic fields with all else remaining constant.

The modified microgel electrophoresis assay or Comet Assay for single DNA-strand breaks, involves extraction of a sample of tissue, washing it several times to remove blood, snipping the tissue with sharp scissors to reduce the sample sizes and further washing to remove blood. Single cell suspensions are mixed with agarose to make a microgel on a slide that is cooled to form a gel. Slides are immersed in an ice-cold lysing solution and then stored in the dark at 4 °C.

DNA is closely associated with protein and RNA. They help to fold the DNA. To release DNA from these bonds, one has to use Proteinase K to digest proteins and RNAase A to digest RNA. Hence in the morning the slides were treated with DNAase-free proteinase K for 2 hr at 37 °C to remove the bound protein from the DNA. They were then places on the horizontal slab of an electrophoretic assembly. An electrophoresis buffer is added and the sample is left for 20 min to allow the DNA to unwind. The buffer includes antioxidants to counter the free radicals produced by electrophoresis.

The electrophoresis was then carried out for 60 minutes with 0.4 V/m, 250 mA. During this process the fluid in the assembly is re-circulated at the rate of about 100 ml/min. The negatively charged segments of DNA migrate down the electric field gradient, forming a comet-like tail, the mass of which is proportional to the amount of damaged DNA material and the electric field gradient and time of exposure.

For DNA double-strand breaks the microgel preparation is the same as above. Slides are then treated with ribonuclease A for 2 hr and then proteinase K for 2 hr. They are then placed in the neutral electrophoresis buffer (pH 9) for 20 mins and then electrophorezed for 1 hr at 0.4 V/cm. For both single- and double- strand assays the sample are stained with an intense florescent dye solution of YOYO-1 and then examined in a vertical florescent microscope.

The proteinase K treatment is vital. It removes the bound protein from the DNA strands. DNA and protein have the opposite charge and so for the electric field to cause migration, the protein must be removed. Four slides were prepared for each animal, two for single and two for double-strand assays. Fifty representative cells were scored off each slide, giving 100 cells scored for each of the single and double-strand DNA breaks. Frequency distributions for the 100 assayed cells are presented, Figure 21, and the comet tail moment calculated.

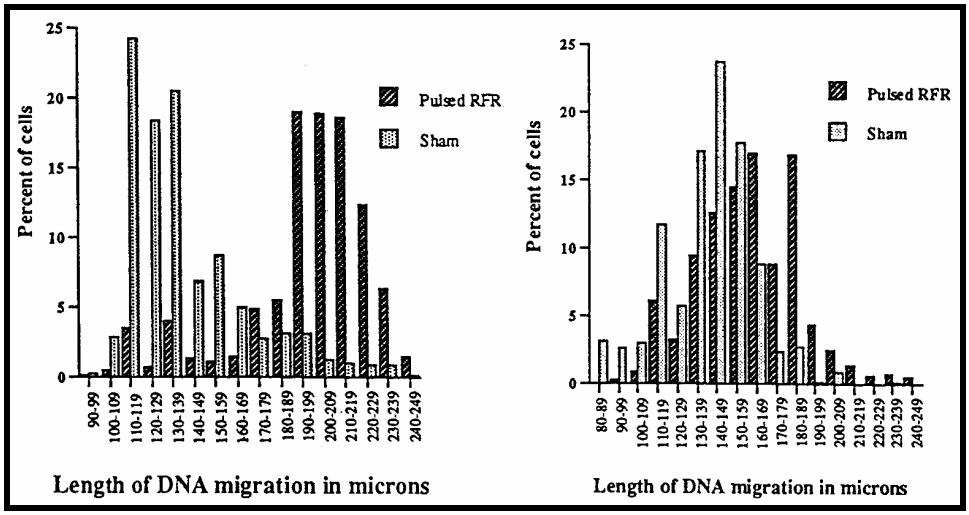

Figure 21: Single- and double-strand DNA breaks frequency distribution for percentage of cells of a given tail length from pulsed RFR and sham exposed brain cells, from 8 animals and 100 cells per animal, Lai and Singh (1996).

Figure 21 clearly shows significant increases in single- and double-strand DNA breaks from the pulsed microwave exposed animal brains compared with the sham exposed

animals. The tail DNA fragments extend out to 250 microns. The Comet tails in the Malyapa et al. assay extend to less than 40 microns. This clearly documents how less sensitive their method is.

Motorola Funded Counter Research on DNA breakage:

Motorola funded Dr Joseph Roti Roti's group at Washington University, St Louis, to replicate the Lai/Singh DNA damage research and to extend it to cell phone frequencies. "Replication" requires the work to be very closely following the method and conditions of the earlier study. While both groups used 2.45 GHz microwaves for exposure, the follow up study used a cell line (C3H/10T1/2) compared to living rats, and they used a very different DNA damage assay based on Olive et al. (1992) not Singh et al. (1994). This follow up study used a much weaker fluorescent stain, an overall weaker electrophoresis field (0.6 V/cm for 25 mins c.f. 0.4 V/cm for 60 mins) and did not use proteinase K to separate the bound protein from the DNA strands. It is therefore understandable why they didn't observe DNA stand breakage from MW exposure.

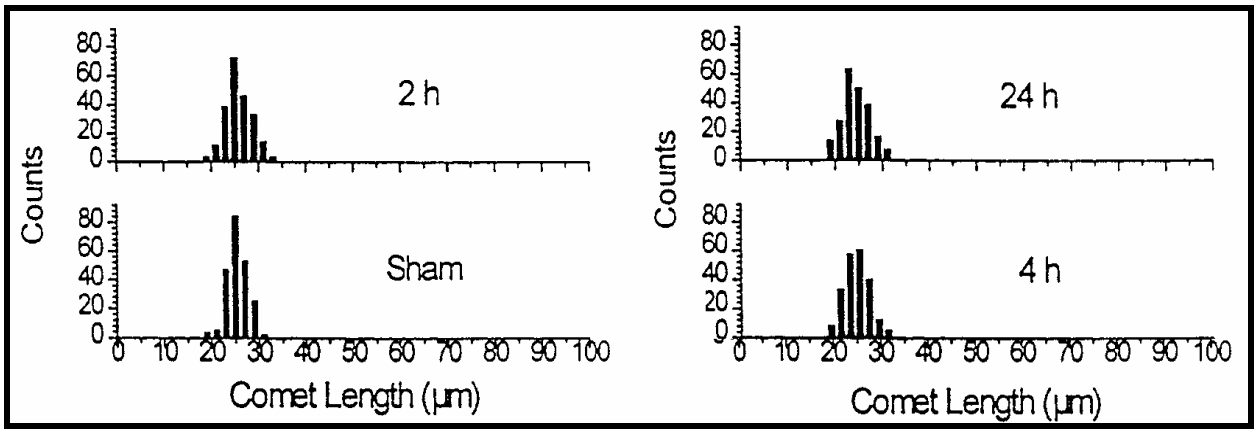

Figure 22: Frequency Distribution of Comet tail lengths for 2.45GHz exposed C3H10T1/2 cells, Malyapa et al. (1998).

Differences between Lai and Singh and Malyapa et al.:

There are five primary differences between the Lai and Singh Comet Assay method derived from Singh at al. (1994) used at the University of Washington and the Comet assay method used at Washington University by Malyapa et al, derived from Olive et al. (1992).

The following factors make the Lai/Singh Assay more sensitive than that of Malyapa et al.:

Complete lysis using highly concentrated salt and two detergents.

The use of proteinase K to remove the positively charges bound protein from the negatively charged DNA stands so that the electrophoresis field produces more migration.

The use of antioxidants during electrophoresis.

Electrophoresis for a longer time to allow longer tails to form in the "Comet". Lai and Singh have 250 micron tails while Malyapa et al. have 40 micro tails.

The use of the YOYO-1 dye. YOYO-1 is 100-fold more sensitive when bound to DNA than propidium iodide.

Hence there are basic practical scientific reasons why Lai and Singh observe EMR-induced DNA strand breaks with RF/MW exposures, whereas Malyapa et al. don't. Two independent laboratories have shown that EMR, including cell phone radiation at extremely low intensities, causes DNA strand breaks. They are Verschaeve et al. (1994) and Phillips et al. (1998), who used the Lai/Singh method.

The Comet Assay and EMR effects:

Drs Lai and Singh have now shown that ELF and RF/MW radiation both cause single and double strand DNA breakage and are associated with free radical and reduced melatonin in living exposed rats. Lai and Singh (1995) observed a dose response increase in Single-strand DNA breakage in the rat's brain and hippocampus that increased significantly after 4 hours, Figure 23.

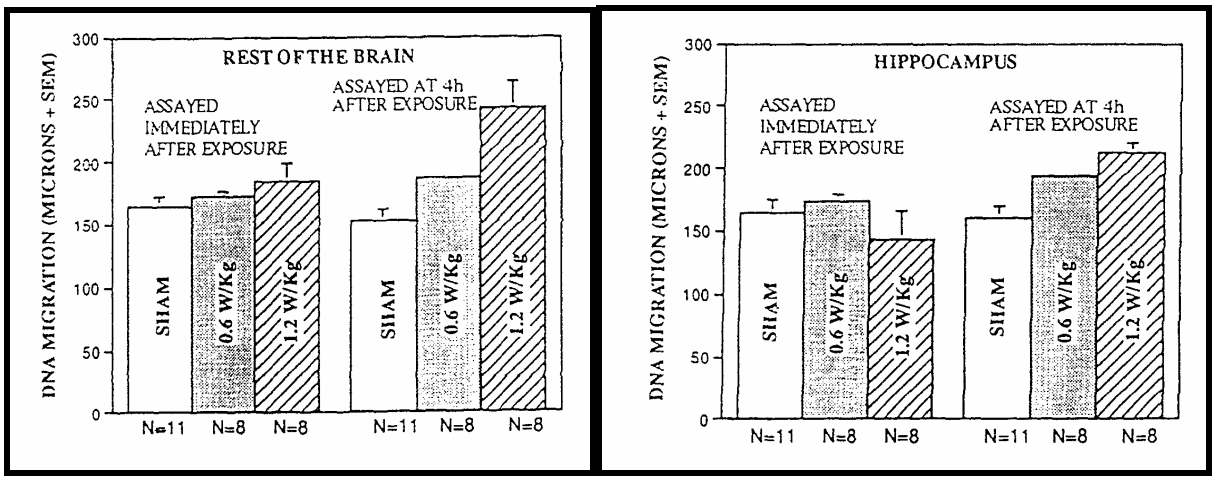

Figure 23: DNA single-strand breakage in cells from the rat brain and hippocampus, immediately after a 2 hr exposure to a whole body SAR of 0.6 and 1.2 W/kg to 2.45 GHz microwave radiation, pulsed at 500 pps. N is the number of rats studied. Lai and Singh (1995).

The increases in DNA single-strand breakage after 4 hrs is highly significant, $p<0.001$ and they show a dose-response relationship.

The assay method was extended to measure DNA double-strand breakage. Lai and Singh (1996) reported that both continuous wave (CW) and pulsed microwaves caused significant ($p<0.01$) increased single-strand DNA breakage, and double-strand breakage, CW, $p<0.05$) and pulsed, $p<0.01$), Figure 24.

This shows that both continuous and pulsed microwaves cause single and double DNA strand breakage, but pulsed microwaves cause more than continuous waves. Hence pulsed cell phone signals and radar signals are highly likely to cause DNA damage. This has been confirmed for radar and chromosome aberrations above and for cell phones by Phillips et al. (1998).

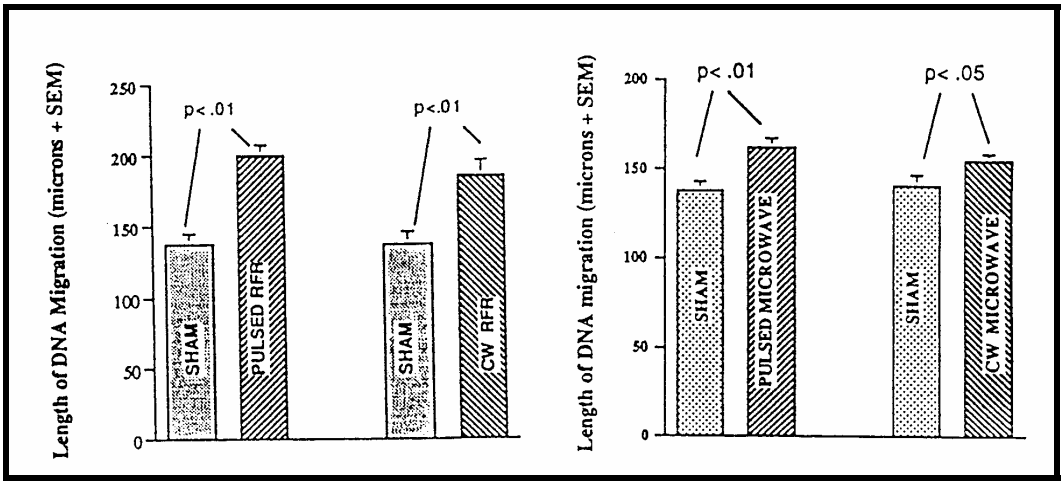

Figure 24: Single-strand (left) and double-strand (right) breaks in brain cells of rat after exposure to pulsed or continuous-wave RFR. Each bar represents data from 8 rats, Lai and Singh (1996).

In the mean time Lai and Singh (1997) investigated the mechanism which is involved with this genotoxic effect of RF/MW radiation. They treated the microwave exposed rats with melatonin and a spin-trap compound (PBN) to determine the role of free radicals. They showed that both melatonin and PBN eliminated the microwave induced DNA damage. Figure 25 shows the effect of melatonin for single- and double- strand DNA breaks and Figure 26 the same for PBN.

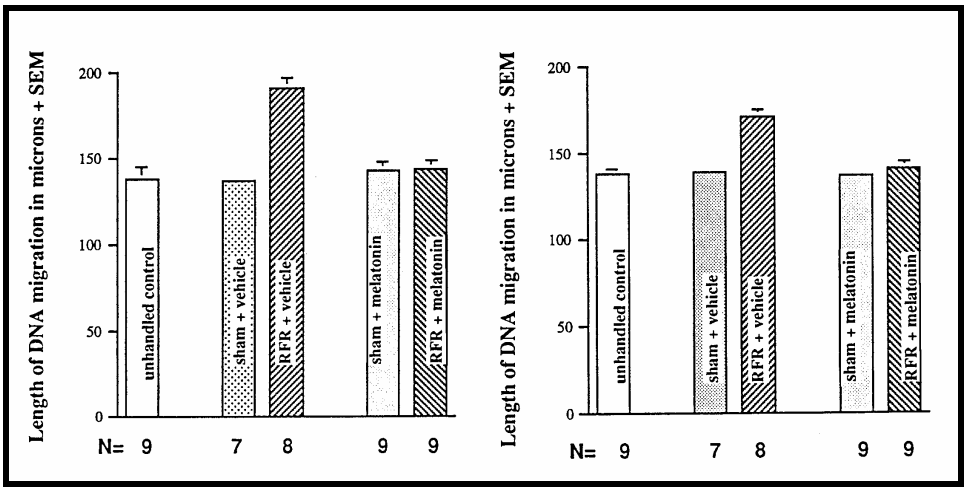

Figure 25: Effect of treatment with melatonin for RFR-induced increase in DNA single-strand (left) and double-strand (right) breaks in rats brain cells. Data was analysed using the one-way ANOVA, which showed a significant treatment effect ($p<0.001$) for both cases. "vehicle" involves injecting with the physiological saline without the active substance. Lai and Singh (1997)

Lai and Singh (1997) conclude that if free radicals are involved in the RFR-induced DNA strand breaks in brain cells, the results of their study could have an important implication of the health effects of RFR exposure. Involvement of free radicals in human diseases, such as cancer and atherosclerosis, have been suggested. Free radicals also play an important role in aging processes, Reiter, (1995). They also point out that both melatonin and PBN can have other actions on cells in the brain that can decrease DNA damage. Therefore further support is necessary to interpret these results.

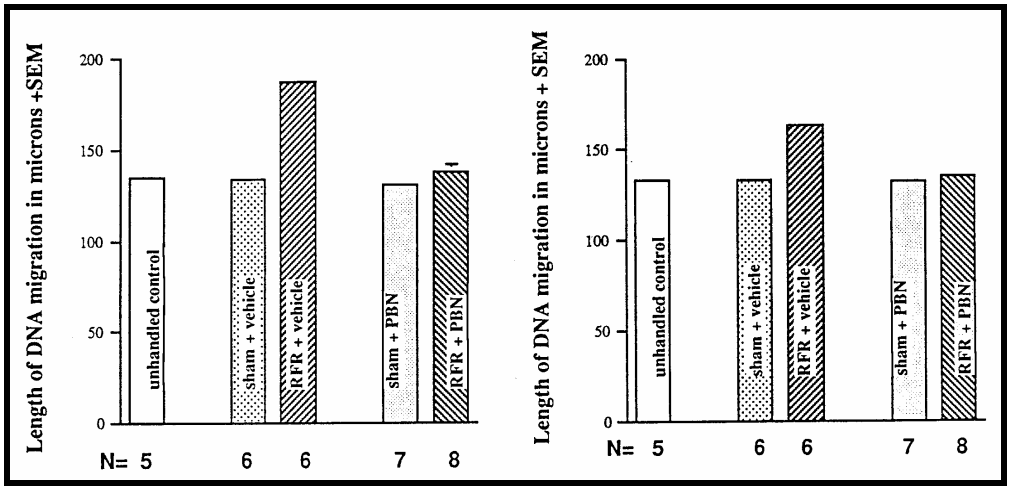

Figure 26: Effect of treatment with PBN for RFR-induced increase in DNA single-strand (left) and double-strand (right) breaks in rats brain cells. Data was analysed using the one-way ANOVA, which showed a significant treatment effect ($p<0.001$) for both cases. "vehicle" involves injecting with the physiological saline without the active substance. Lai and Singh (1997).

Phelan et al. (1992) exposed B-16 melanoma cell line to pulsed 2.45 GHz, 100 pps, 1hr exposure SAR = 0.2 W/kg. This resulted in changes of membrane ordering. Their data indicated that a significant, specific alteration of the cell-membrane ordering followed microwave exposure and that the alteration was due at least part, to the generation of oxygen radicals. Hence there is independent support for the generation of free radicals by microwaves, as well as the Lai/Singh evidence that PBN and Melatonin reduce the RFR induced DNA damage.

Two other laboratories have recorded RF/MW produced significant DNA stands breaks. Verschave et al. (1994), who used a GSM cell phone signal to expose human and rat peripheral blood lymphocytes, found significantly increased strand breaks at high, but non-thermal exposure levels.

Cellphone Radiation causally breaks DNA:

Phillips et al. (1998) exposed Molt-4 T-lymphoblastoid cells the a range of cell phone radiation in the SAR range 0.0024 W/kg to 0.026 W/kg for both iDEN and TDMA signals. Using the basic equations, these SARs at the 813-836 MHz range [SAR = $\sigma E^2/2\rho$, σ=1 S/m, ρ=920 kg/m^3, and S = $E^2/3.77$ μW/cm^2, E: the electric field gradient in V/m and S the exposure in μW/cm^2]. Thus S = 488 SAR. This gives 1.2 to 12.7μW/cm^2. A 2 hr exposure to these low levels of cell phone radiation extremely significantly increased ($p<0.0001$) or decreased ($p<0.0001$) the DNA damage. Decreased DNA damage is evidence of increased repair that is evidence of damage, Meltz (1995). These are extremely significant at the causal level. It also shows that even at 0.1μW/cm^2 the DNA damage is likely to be extremely significant. This is so significant that it is causal.

RF/MW DNA breakage Conclusions:

Hence RF/MW radiation has been confirmed to enhance DNA damage under RF/MW exposure from radar-like and cell phone exposures, including an exposure level which is 0.22% of the ICNIRP guideline.

ELF Exposure and DNA strand breakage:

Four independent laboratories have also published data on ELF induced DNA strand breaks confirming that ELF EMR damages DNA strands; Lai and Singh (1997a), Svedenstal et al. (1998), Phillips et al. (1998a), and Ahuja et al. (1997). Lai and Singh (1997a) also demonstrate the involvement of free radicals and the protective effect of melatonin. With the evidence above that EMR reduces melatonin this confirms that reduced melatonin causes higher concentrations of free radicals which produce more DNA strand breaks from EMR exposure from ELF to RF/MW frequencies. Increased DNA strand breaks will result in increased chromosome aberrations.

Multiple evidence from independent laboratories established that EMR from ELF to RF/MW causes DNA single- and double-strand breaks at very low, non-thermal exposure levels. This extends and confirms the genotoxic evidence from chromosome aberration studies.

EMR Altered Gene Activity

There is also evidence that EMR not only can damage chromosomes and DNA strands, but it is observed to alter cellular calcium ions and the activity levels of proto oncogenes (cancer genes).

Blackman (1990) concluded that there was overwhelming evidence that EMR can alter normal calcium ion homeostasis and lead to changes in the response of biological systems to their environment. On of these changes is altered gene transcription and expression. The lowest published exposure level associated with significant EMR-induced alteration of cellular calcium ions occur is reported by Schwartz et al. (1990). It was 0.00015 W/kg in a 30 min exposure to a 240 MHz signal modulated at 16 Hz. The medium was frog hearts. This is equivalent to an exposure level of about 0.08 $\mu W/cm^2$.

Calcium ion fluxes occur in “windows” of exposure parameter combinations. Two studies associate EMR exposure alteration of gene transcription with exposure windows. Litovitz et al. (1990) identified amplitude (intensity) windows, and Wei et al. (1990) frequency windows in the range 15 to 150 Hz. They observed a peak effect in c-myc gene transcription at 45 Hz. Liburdy et al. (1993) show that c-myc induction occurs in a direct sequence from calcium ion influx. Increased c-myc gene transcripts by 50/60 Hz fields has also been observed, Goodman et al. (1989, 1992) and Lin et al. (1994). Phillips et al. (1992, 1993) observed time-dependent changes in the transcription of c-fos, c-jun, c-myc and protein kinase C, from 60 Hz exposure and a linear reduction in ras p21 expression by a 72 Hz signal. 50/60 Hz signals altered c-jun and c-fos gene expression as observed by and Lagroye and Poncy (1998) and c-fos expression by Rao and Henderson (1996) and Campbell-Beachler et al. (1998). The ppSom gene is very important in human neurological disorders, and is regulated by calcium ions Capone, Choi and Vertifuille (1998).

Cell phone radiation (836.55 MHz) significantly altered c-jun transcript levels, Ivaschuk et al. (1997). Cell phone radiation significantly enhances the proto oncogene c-fos activity in C3H 10T 1/2 cells, from a 40 % ($p=0.04$) increase from a digital cell phone and a 2-fold increase ($p=0.001$) from an analogue cell phone, Goswami et al. (1999).

Hence proto oncogene activity is altered and enhanced in multiple independent experiments from ELF and RF/MW exposure, including cell phone radiation.

Immune system impairment by EMR

Impairment of the immune system is related to calcium ion efflux, Walleczek (1992) and to reduced melatonin, Reiter and Robinson (1995). Cossarizza et al. (1993) showed that ELF fields increased both the spontaneous and PHA and TPA- induced production of interleukin-1 and IL-6 in human peripheral blood. Rats exposed to microwaves showed a significant reduction in splenic activity of natural killer (NK) cells, Nakamura et al. (1997).

Dmoch and Moszczynski (1998) found that microwave exposed workers had decreased NK cells and a lower value of the T-helper/T-suppressor ratio was found. Moszczynski et al. (1999) observed increased IgG and IgA and decreased lymphocytes and T8 cells in TV signal exposed workers. Quan et al. (1992) showed that microwave heating of human breast milk highly significantly suppressed the specific immune system factors for E.Coli bacteria compared with conventional heating. Chronic, 25 year, exposure to an extremely low intensity (<0.1μW/cm^2) 156-162 MHz, 24.4 Hz pulse frequency, radar signal in Latvia produced significant alterations in the immune system factors of exposed villagers, Bruvere et al. (1998).

EMR Reduces Melatonin in Animals and People

DNA strand breaks, Chromosome Aberrations, impaired immune system competence and many other biological and health effects, are caused by reduced melatonin, Reiter and Robinson (1995). Light-at-night and electromagnetic radiation, are proven to reduce melatonin and hence pose significant adverse health effects.

The evidence for EMR reduction of melatonin is summarized here. Rosen, Barber and Lyle (1998) state that seven different laboratories have reported suppression of nighttime rise in pineal melatonin production in laboratory animals. They show that a 50 μT, 60 Hz field with a 0.06μT DC field, over 10 experiments, averages a 46% reduction in melatonin production from pinealocytes. Stark et al. (1997) observed a significant increase in salival melatonin in a group of 5 cows when the short-wave radio transmitter at Schwarzenberg, Switzerland, was turned off for three days, compared to 5 cows that had much lower RF exposure. Hence there are now nine independent observations of melatonin reduction in animals from ELF and RF exposure.

Fifteen studies from show that ELF and RF/MW exposure reduces melatonin and enhances serotonin in people. Evidence that EMR reduced melatonin in human beings commenced with Wang (1989) who found that workers who were more highly exposed to RF/MW had a dose-response increase in serotonin, and hence indicates a dose-response reduction in melatonin. Fourteen studies have observed significant EMR associated melatonin reduction in humans. They involve a wide range of exposure situations. This includes 16.7 Hz fields, Pfluger et al. (1996); 50/60 Hz fields, Wilson et al. (1990), Graham et al. (1994), Wood et al. (1998), Karasek et al. (1998), Burch et al. (1997, 1998, 1999a, 2000), Juutilainen et al. (2000) and Graham et al. (2000a); combination of 60 Hz fields and cell phone use, Burch et al. (1997,1999a); VDTs ELF/RF exposures, Arnetz et al. (1996), and a combination of occupational 60Hz exposure and increased geomagnetic activity around 30nT, Burch et al. (1999b).

The fourteenth human melatonin reduction study is from 6.1-21.8 MHz SW RF exposure as reported during the shutting down process of the Schwarzenburg shortwave radio tower, Professor Theo Abelin (seminar and pers.comm.). Urinary melatonin levels were monitored prior to and following the closing down of the Schwarzenburg short wave radio transmitter. This showed a significant rise in melatonin after the signal was turned off.

Fifteen studies is sufficient to establish that EMR reduces melatonin in people from exposures across the EMR spectrum, and at extremely low mean exposure levels.

Genotoxicity Conclusions:

There is more than sufficient evidence of chromosome aberrations, DNA strand breakage altered oncogene activity and neoplastic transformation of cells to conclude that EMR across the spectrum from ELF to RF/MW is genotoxic. This is independently confirmed by the established biological mechanisms of calcium ion efflux and melatonin reduction.

This is also totally independent of over a hundred occupational groups showing elevated cancer from EMR exposure, scores showing significantly to extremely significantly elevated cancer incidence and mortality, and dozens of dose-response relationships.

Epidemiological Evidence of Cancer:

WHO 1993 reviews six cancer related studies and ICNIRP 1998 cites only 13 claimed cancer related studies. Two of them aren't so that leaves only 11 studies. Two of the primary studies they use to claim that RF/MW does not cause cancer in humans are Lilienfeld et al. (1978), the U.S. Embassy in Moscow Study and the Korean War Study, Robinette et al. (1980). Both of the conclusions of these studies are suspect because the conflict with the data contained in the papers and reports. Blood samples from the Moscow Study show significant chromosome aberrations. The following is a summary table from the tables in Lilienfeld et al.

U.S. Embassy in Moscow Study:

The all cause mortality rate for Moscow males as 0.42 (95%CI: 0.3-0.6) and for females 1.1 (95%CI: 0.5-1.9). Hence males, primarily State Department employees, were much healthier and females were as healthy as the average U.S. residents. This is a good example of the "healthy worker" effect. State Department selection procedures rule out a range of unhealthy people and favour healthy people.

The U.S. Embassy was chronically exposed to a radar signal from 1953 to 1976+. Up to 1975 there was one radar pointed at the upper levels of the West Façade. The measured intensities on the outside walls gave a peak reading of 5 $\mu W/cm^2$, over 9 hours/day. This averages 1.9$\mu W/cm^2$, on the outside wall. It was far less inside.

From our knowledge of the effects of Geomagnetic Activity and ELF studies, we would expect cardiac and neurological effects, and, with reduced melatonin, many other illnesses. Two of the most significant results were the increase in illness in a significant dose-response manner with years of service in Moscow, Table 2. This included cardiac symptoms (Vascular System). Generally two or three comparisons are made. The rate of

health effects were compared with the U.S. population of similar ages, with comparative Eastern European Embassy people, and some exposed vs non-exposed groups.

Table 2: Sickness rates increased in Moscow with years of service: (Table 6.18)

	Under 2 yrs	2-3 years	4 + years	p-value for trend
Number of people	316	455	45	
Person-years	3709	5570	568	
Male Conditions (%)				
Present Health Summary	5.4	9.7	16.2	0.05
Arthritis/rheumatism	4.3	6.5	8.8	0.02
Back Pain	4.0	7.7	11.8	0.04
Ear problems	3.8	5.6	14.7	0.02
Vascular system	0.8	2.7	11.8	0.004
Skin & Lymphatic	9.4	12.2	28.0	0.02
Female Conditions (%)				
Vaginal discharge	4.2	13.8	17.5	0.04

The sickness rates increased independent of the age of arrival and much faster than the influence of aging.

The second was the neurological symptoms that were significantly elevated in female employees and highly significantly elevated in male employees. This difference between men and women is probably a consequence of sample size. "Comparison" refers to other Eastern European Embassies.

These symptoms are consistent with the "Microwave Syndrome" of the "Radiofrequency Radiation Sickness", Johnson-Liakouris (1998). Mild et al. (1998) identified significant dose-response relationships for the following symptoms from the use of mobile phones: Memory Loss, Difficulty in Concentrating, Headache, and Fatigue.

Table 3: Neurological Symptoms per 1000 p-y, Male employees: (Table 6.31)

	Moscow	Comparison	RR	p-value
Depression	1.3	0.73	1.78	0.004
Migraine	1.8	0.97	1.86	
Lassitude	1.2	0.78	1.54	
Irritability	1.3	0.66	1.97	0.009
Nervous Disorders	1.5	0.64	2.34	
Difficulty in Concentrating	1.4	0.52	2.96	0.001
Memory Loss	1.6	0.50	3.20	0.008
Dizziness	1.2	0.85	1.41	
Finger Tremor	1.3	0.71	1.83	
Insomnia	1.1	0.90	1.22	
Neurosis	1.3	0.76	1.71	

Hence it is now shown and known that RF/MW exposure from extremely low but chronic exposure over many years, occupational exposure and cell phone use for minutes/day all produce significant and consistent neurological symptoms. The Risk Ratios for other symptoms were quite large but they were not quite significant because of the very small sample numbers.

Chromosome Damage in blood samples:

Table 4: Blood samples showed a high proportion of the staff had significantly altered red and white blood cell counts and well above average chromosome aberrations (CA). The CA data is set out in Goldsmith (1997), i.e.

Mutagenic Level	Designator	Subjects, No.
5	Extreme	0
4	Severe	6
3.5	Intermediate	5
3	Moderate	7
2.5	Intermediate	5
2	Questionable	5
1	Normal	6

Patients with mutagenic level of 3 and above were advised not to reproduce until 6 months after somatic levels had returned to 2 or 1. This warning applied to 68 % of the patients in this sample. Staff who had elevated chromosome aberration rates were advised not to have children for until six months after they had returned to near normal.

Because of the vulnerability of children it is interesting to consider the observed health effects on childhood dependents.

Table 5: Congenital Malformations of children after the first tour:

Conditions	Moscow SMBR	Comparison SMBR	RR	Number of children
Leukaemia and cancer	1.2	0.84	1.43	1
Blood Disorders	1.7	0.42	4.05	7
Mental, Nervous Cond^{n}.	1.8	0.36	5.0	8
Behavioural Problems	1.4	0.68	2.06	7
Chronic Disease	1.1	0.88	1.25	7

A survey of cancer mortality rates is summarized in Table 6. This shows that despite the extremely small sample size and the very significant exposure dilution in the years between residence in Moscow and the survey results, there are highly elevated and significantly elevated rates of mortality from cancer Lilienfeld et al. shows significantly increases chromosome aberration and cancer. This was recently also found in mice, Vijayalaxmi et al. (1997).

The dominant cancers are brain tumor and leukaemia and reproductive organ cancer. But this study, like the Korean War Study, confirms that extremely low level chronic microwave exposure is associated which very significant increases in illness and mortality in organs across the whole body, consistent with widespread cellular chromosome damage.

Significantly elevated chromosome aberrations were measured in this case, Table 13, as well as significant alterations in white and red blood cell counts, Jacobson (1969). This would also be the expected result from reduced melatonin.

Table 6: Cancer Mortality Rates:

Male employees (Table 6.37)	Moscow SMBR	Comparison SMBR	RR
Skin Cancer	1.5	0.69	2.17
Benign Neoplasms	1.4	0.75	1.87
Female employees (Table 6.38)			
Malignant Neoplasm (Excl. skin)	1.7	0.63	2.86 (p=0.06)

Adult Dependents: (Tables 7.12, 7.13)

	Obs.	Exp	SMR	(95%CI)
Live-in				
All malignant Neoplasms	5	1.5	3.3	(1.1-7.7)
Digestive Organs Cancer	1	0.26	3.8	(0.1-21.2)
Pancreas Cancer	1	0.03	33.3	(0.8-185)
Breast Cancer	1	0.4	2.5	(0.1-13.9)
Ovarian Cancer			3.0	
Multiple Myeloma			1.5	
Arteriolosclerotic Heart Disease	2	0.59	3.4	(0.4-12.3)
Live-out				
All malignant Neoplasms	7	3	2.3	(0.9-4.7)
Brain tumor	2	0.1	20.0	(2.4-72.2)
Lung cancer	1	0.44	2.3	(0.4-93)
All Accidents	4	1	4.0	(1.1-10.2)
Suicide	1	0.36	2.8	0.1-15.6)
Children Living In (Table 7.16)				
All Malignant Neoplasms	2	0.5	3.8	(0.5-13.7)
Leukaemia	1	0.2	5.3	(0.1-29.5)
Suicide	1	0.29	3.4	(0.0-1.6)
Children Living out				
All Malignant Neoplasms	2	0.83	2.4	(0.3-8.7)
Leukaemia	1	0.3	3.4	(0.1-18.9)
Suicide	1	0.3	3.3	(0.1-18.4)

Overall summaries of morbidity experience for dependents were carried out.

For adult dependents it was concluded: "Thus, those who lived in Moscow had more conditions with higher mortality ratios than other groups, particularly those who had not lived in any of these posts." Individual conditions were rather too small to achieve statistical significance but the overall effect, while small, is greater in Moscow.

Female employees, Table 6.38, experienced 22 malignant neoplasms in 2557 PY compared to 17 in 4662 PY, i.e.8.6 /1000 PY vs 3.65 / 1000 PY, giving RR = 2.36, 95%CI: 1.25-4.44. This is a highly significant result.

The overall summary for dependent children showed: "The dependent children who has definitely lived in Moscow had more conditions with higher SMBRs (Specific Morbidity Ratios) in two out of three comparisons; however these differences were minimal." Thus the children showed a similar pattern to the adults, of a slightly higher mortality pattern in Moscow.

The Health History Questionnaire (HHQ) results for male employees, when summarized concluded, p156: "There was a clear pattern of a higher frequency of symptoms reported by the Moscow Group than was reported by the Comparison Group. For males of the 20 categories of symptoms, 17 of the SMBRs were higher in the Moscow Group and 4 of them (depression, irritability, loss of appetite and difficulty concentrating) were statistically significantly different.

Attempts to classify people with the Moscow Embassy met with mixed success and mixed results. Comparisons with the Moscow and Comparison Groups yields a consistent pattern of increased morbidity in Moscow. Female cancer and male neurological symptoms were significantly elevated.

Report conclusions challenged:

It is stated by both Bradford Hill (1965) and Goldsmith (1992) that elevated Odds and Risk Ratios are also relevant to the public health protection basis in epidemiology,

Professor Goldsmith, an eminent epidemiologist, was closely associated with the staff affected by the chronic radar exposure of the U.S. Embassy in Moscow and obtained information through the Official Information Act. This included the blood test results and minutes of meetings which record the fact that the State Department case officer, Dr Herbert Pollack, changed the conclusions of the final report compared with the draft report, to state that no effects could be associated with the radar exposure, Goldsmith (1997). The data and Dr Goldsmith show that this is not true. After reviewing this data Dr Goldsmith, Goldsmith (1995), referring to a “recent draft of criteria for health protection” which claims: “No effect on life span or cause of death of 1,800 employees and 3000 dependents of the U.S. Embassy personnel”, states:

> **“To ignore these findings on the basis of “No effect on life span or cause of death” in setting human exposure standards is wrong. In the first place the criteria are two narrow; mortality is not the only relevant end-point. The positive or 'findings for concern' are ignored. Increased cancer incidence among dependents is a nontrivial endpoint.”**

This body cited was the predecessor of ICNIRP.

Allowing for the fact that several years had elapsed between the exposure and the health survey, allowing the opportunity for exposure dilution, the relatively short follow-up period and allowing for the very small sample sizes, these results are remarkable.

A highly remarkable result is the dose-response relationship for a range of sicknesses, Table 10. The results must be very highly significant to survive the exposure dilution effect with the disease gradient intact and statistically significant. As with Robinette et al. (1980), the data presented in the Lilienfeld contract report is contrary to that stated in the report's stated (an altered) conclusions. Despite the small numbers, the lack of long latency period and dilutionary factors, the Lilienfeld data shows a significant increases in:

- Cardiac symptoms
- Neurological and psychological symptoms
- Altered blood cell counts
- Increased chromosome aberrations, and
- Elevated cancer in children and adults
- Sickness increasing in a dose-response manner with years of residence.

These symptoms are associated with chronic exposure to very low intensity pulsed microwaves in the range < 0.04 to 0.2μW/cm^2.

In a sense too, the fact that the State Department case officer, Dr Herbert Pollack, altered the conclusions, attests to the significance of this study. The results would have been embarrassing to the U.S. Government, both in terms of workers compensation and in terms of the validity of the U.S. exposure standard.

That the WHO (Dr Repacholi) and ICNIRP still claim that this study showed no adverse health effects strongly challenges their professionalism and objectivity. The data strongly challenges the altered conclusions. The effects are highly consistent with other studies.

The Korean War Study:

This is the second major study that is claimed by WHO and ICNIRP to show no adverse health effects from radar exposure. Robinette et al. (1980) studied the health effects of radar exposed naval technical personnel who had served on ships during the Korean War. This illustrates one of the major problems with cancer and morbidity health studies. The desirable situation is to have clearly separated exposure groups so that a highly exposed group is able to be compared with a non-exposure or very low exposure group. It is also very highly desirable to identify an intermediate group to determine if there is a dose-response relationship.

It takes decades for most cancers to develop after there initiation by a carcinogenic agent. In the time between the exposure and the health effects survey, a highly variable and probably random exposure regime will influence the subjects in the mean time, significantly reducing the dichotomy and, unless the effect is very strong, eliminating any initial exposure gradient. This is called exposure dilution and it significantly reduced the chances of observing any effects.

In this study the definition of low exposure groups was those operating radios and radars. The high exposure group was those repairing and maintaining the radios and radars.

Three occupational groups were placed in the highly exposed group, Electronics Technician (ET), Fire control Technician (FT) and Aviation Electronics Technician (AT). When a 5% sample of servicemen in these three groups were surveyed using a job-matrix exposure survey, they found a gradient in mean exposure with ET being low, FT intermediate and AT being high.

The individuals who were surveyed were used for a dose-response analysis, they were shown to have a significant dose-response increase in Total Mortality and Respiratory Cancer as a function of exposure level as assessed by the Hazard Number. Figure 27 shows the dose-response relationships for these mortalities with the lowest exposure range used as a reference with RR=1.0.

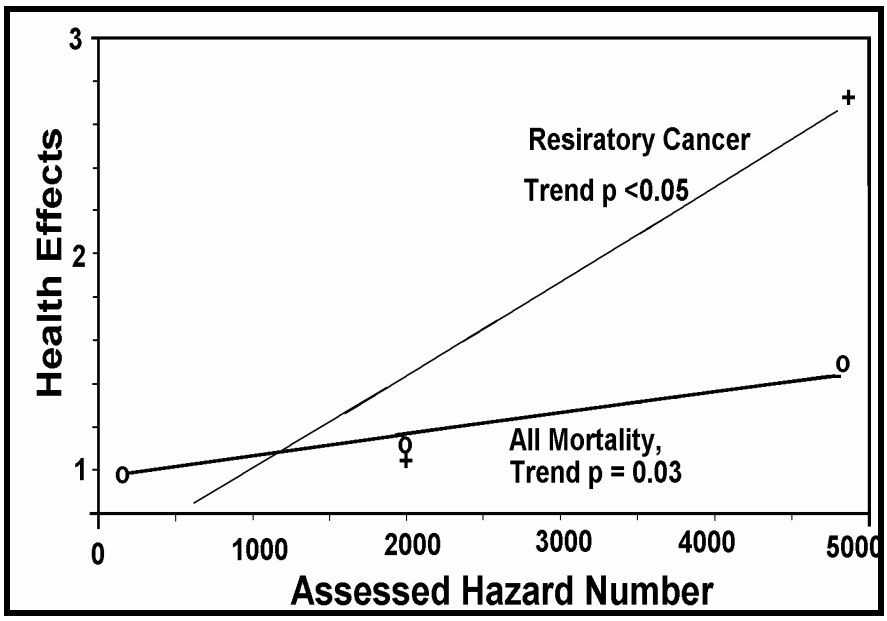

Figure 27: Dose-response relationships of mortality from all causes and respiratory cancer for radar exposure assessed personnel, Robinette et al. (1980).

This is an amazing result given the dilution factors. Several other symptoms showed dose-response increases, including Malignant Neoplasms, Lymphatic and Hematopoietic cancers, other malignant neoplasms and disease of the circulatory system.

Using the originally identified occupational groups a problem arises. The authors, on Navy advice, have placed the high mortality group, Aviation Electrician's Mate (AE), which is clearly a repairers' group, in the operators' group. This is the smallest group by far and so the simplest solution is to remove it altogether. Taking the Radarman (RD) and Radioman (RM) as the low exposure group, ET + FT as the intermediate exposure group and AT as the high exposure group, then data in Robinette et al.'s table 5, for mortality from all causes yields Figure 28.

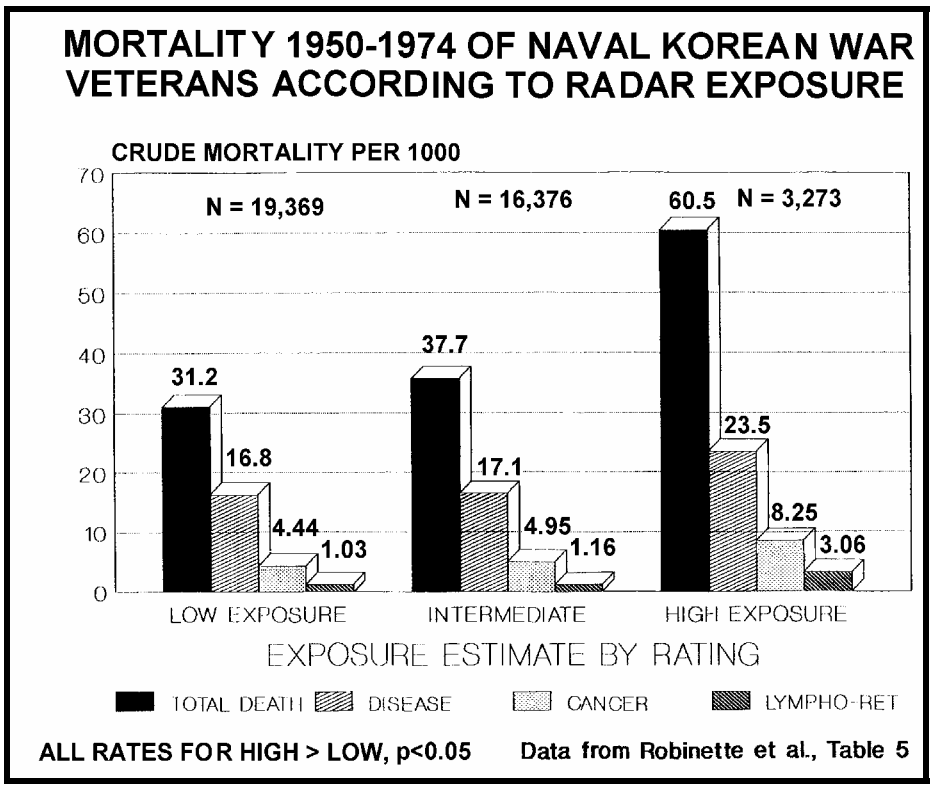

Figure 28: Naval occupations grouped by exposure category, showing dose response increases in mortality for all mortality, all disease, cancer and Lymphatic/Leukaemia. Low exposure (RM+RD), Intermediate exposure ET+FT), High exposure (AT).

Grouping occupational groups according to exposure levels also reveals dose-response increases for Total Death, All Disease, All Cancer and Lymphatic/hematopoietic Cancer, Figure 28. This strengthens the 5 % group survey result. It shows that radar exposure is strongly correlated with increased total mortality, mortality from disease, cancer and lymphatic/hematopoietic cancer, Table 7.

Table 7: Number of deaths from disease and mortality ratios by Hazard Number: US enlisted Naval personnel exposed to microwave radiation during the Korean War period, from Table 9, Robinette et al. (1980). The Rate Ratio is calculated as the ratio of the Mortality ratio for Hazard Number 5001+ exposure and 0 Hazard Number exposure.

Cause of Death		Hazard Number			Trend		
	No.	0	1-5000	5001+	p-value	RR	95%CI
All diseases	309	0.82	0.91	1.23	0.03	1.50	1.08-2.08
Malignant Neoplasms	96	0.99	0.90	1.44	N.S.	1.45	0.83-2.52
Digestive Organs	20	1.49	1.14	0.78	N.S.	0.52	0.13-2.08
Respiratory Tract	24	0.82	0.86	2.20	<0.05	2.68	0.84-8.55
Lymphatic and hematopoietic System	26	1.09	1.04	1.64	N.S.	1.50	0.52-4.32
Other Malignant neoplasms	26	0.78	0.70	1.17	N.S.	1.50	0.52-4.32
Disease of Circulatory System	150	0.94	0.83	1.17	N.S.	1.24	0.79-1.94
Other Disease	63	0.30	1.13	1.08	N.S.	3.60	1.14-9.20

Given the exposure dilution factors, all but digestive organs would probably have RR>2 and be significantly be increased. This small sample analysis shows a significant dose response trend for mortality from all diseases ($p=0.03$) and for Respiratory Cancer ($p<0.05$). This is remarkable given the exposure dilution. The analysis also shows that for every disease cause but one there is an elevated risk of mortality due to a range of cancers, Circulatory Disease and Other Disease.

The mean Hazard Number for each group is calculated using a mean hazard number of 0, 1000, 3500 and 6000 for the defined ranges. The mean exposure estimate also shows a gradient and suggests that the best dichotomy will be achieved by comparing AT as a high exposure group to ET as a low exposure group. This was not done by Robinette et al. who preferred to compare ET with the FT and AT groups combined (FT+AT). This maintains larger numbers in the high exposure group by reduces the exposure separation.

The mortality dose-response gradient persists when the total mortality rate is calculated for the ET, FT and AT groups: MR (ET) = 1.0; MR(FT) = 1.29; and MR(AT) = 1.79.

Having identified that the FT and AT groups had higher hazard numbers than the ET group, Robinette et al. combined FT + AT and compared their mortality rates with ET, Table 8. Table 8 shows elevated mortality rates compared with the ET group, for all causes of death listed. The text records that they are significantly elevated for All Disease ($p<0.01$) and Other Diseases ($p<0.01$).

Table 8: Mortality rates, Risk Ratios and Confidence Intervals between the ET group and AT group of US enlisted personnel exposed to microwave radiation during the Korean War.

Cause of death	No.(FT+AT)	ET	FT+AT	RR	95%CI
All diseases	140	0.83	1.19	1.43	1.14-1.79
Malignant Neoplasms	40	0.95	1.18	1.24	0.83-1.86
Digestive Organs	8	1.10	1.19	1.08	0.44-2.65
Respiratory Tract	9	1.13	1.15	1.02	0.45-2.33
Lymphatic and Hematopoietic System	11	1.06	1.40	1.32	0.61-2.87
Other malignant neoplasms	12	0.68	1.06	1.56	0.72-3.37
Diseases of the Circulatory System	64	0.85	1.08	1.27	0.92-1.75
Other disease	36	0.61	1.46	2.39	1.45-3.94

Their Table 5 sets out the mortality data by cause of death for each occupational group, giving the opportunity to compare AT rates with ET rates of mortality. The results are shown in Table 6. In Table 5 where exposures are more dichotomized, mortality due to Malignant Neoplasms and Lymphatic/Hematopoietic cancers are both significantly elevated but when FT and AT are combined these results are no longer significantly different. It is interesting too that in the dose-response analysis using the individual's hazard number, respiratory cancer shows a significant trend, but in these occupational group comparisons this cancer is elevated but not significantly elevated. The comparisons between Tables 9 and 10 clearly show the effect of dilution through combining the FT and AT groups.

Table 9: Mortality Incidence per 1000 and Risk Ratio AT/(RD+RM) as an indication of the high exposure (AT) to low exposure (RD+RM) difference.

	Exposure				
	Low	High	Risk Ratio	95 % CI	p-value
Causes of Death					
All Deaths	31.9	60.5	1.94	1.65 - 2.28	<0.00001
Accidental Death	10.0	29.6	2.97	2.33 - 3.80	<0.00001
Motor Vehicle Death	6.5	6.1	0.95	0.59 - 1.52	-
Suicide, Homicide, Trauma	3.8	6.1	1.60	0.98 - 2.62	0.06
Suicide	2.5	2.8	1.11	0.54 - 2.26	-
All Diseases	16.8	23.5	1.40	1.09 - 1.79	0.007
Malignant Neoplasms	4.4	8.3	1.86	1.21 - 2.86	0.004
Digestive and Peritoneum	0.8	1.2	2.59	1.07 - 6.29	0.03
Respiratory	0.8	2.1	1.75	0.72 - 4.25	-
Eye, Brain, CNS FT/(RD+RM)	0.4	0.9	2.54	0.66 - 9.81	-
Skin	0.3	0.6	1.97	0.40 - 9.77	-
Lymphatic and Hematopoietic	1.0	3.1	2.96	1.39 - 6.32	0.003
Circulatory System Disease	8.8	9.5	1.08	0.74 - 1.58	-
Digestive System Disease	1.2	2.8	2.22	1.03 - 4.77	0.03
Other Diseases	2.1	2.8	1.30	0.63 - 2.67	-

This shows elevated Risk Ratios for all causes of death except motor vehicle and suicide. Highly to extremely significant increases in mortality were found for All Diseases, Malignant Neoplasms, Cancer of the Digestive and Peritoneum systems, Lymphatic and Hematopoietic cancer and from diseases of the Digestive System. Extremely significant increases were found for All Causes of death and Accidental Death.

Morbidity Data:

Robinette et al. obtained two morbidity data sets. The first was from the periods 1952-54 and 1956-59 for admissions to naval hospitals. This is very close to the period of exposure and allows little time for cancers to develop. The second data set was from Veterans' Administration Hospitals for the period 1963-76.

For the immediate post-war data set the following significant increases in sickness were identified by Robinette et al.:

Diseases of the ears, nose and throat ($p<0.01$),
Acute respiratory disease ($p<0.01$),
Other respiratory disease ($p<0.02$),
Diseases of the urinary and male genital organs ($p<0.05$), and
Accidents, poisonings and violence ($p<0.001$).

Table 10: Number of hospitalizations and hospitalization rates per 10,000 per year, in VA hospitals, 1963 -1976, by diagnosis and exposure class: US enlisted Naval personnel exposed to microwave radiation during the Korean War period. The significance p-value is calculate from the Mantel-Haenszel Chi-squared estimate.

					High exposures				
VA diagnostic class	Total		ET		FT + AT				
	No.	Rate	No.	Rate	No.	Rate	RR	95% CI	p-value
Infective, parasitic	42	1.5	24	1.3	18	1.9	1.46	0.79-2.69	0.26
Neoplasms, malignant	34	1.2	17	1.0	17	1.8	1.80	0.92-3.53	0.04
Neoplasms, other	26	0.9	9	0.5	17	1.8	3.60	1.60-8.08	<0.001
Allergic, endocrine system, metabolic and nutritional dis.	77	2.8	41	2.3	36	3.8	1.65	1.05-2.58	0.01
Blood, blood-forming organs	17	0.6	5	0.3	12	1.3	4.33	1.53-12.3	0.001
Alcoholism	105	3.8	45	2.5	60	6.3	2.52	1.71-3.71	<0.001
Other mental disorders	276	10.1	166	9.3	110	11.6	1.25	0.98-1.58	0.02
Nervous system, sense org.	106	3.9	58	3.2	48	5.1	1.59	1.08-2.33	0.009
Circulatory	123	4.5	68	3.8	55	5.8	1.53	1.07-2.18	0.007
Respiratory	80	2.9	43	2.4	37	3.9	1.63	1.05-2.53	0.014
Digestive	255	9.3	132	7.4	123	13.0	1.76	1.38-2.25	<0.001
Genitourinary	82	3.0	45	2.5	37	3.9	1.56	1.01-2.41	0.02
Skin, cellular	61	2.2	33	1.8	28	2.9	1.61	0.97-2.66	0.04
Bones, organs of movement	80	2.9	36	2.0	44	4.6	2.30	1.48-3.57	<0.001
Trauma	108	3.9	53	3.0	55	5.8	1.93	1.32-2.81	<0.001
Symptoms, ill-defined cond., special exams and other	151	5.5	85	4.8	66	6.9	1.44	1.04-1.99	0.007
Person-years (1000)	27.39		17.89		9.50				

Table 10 gives a more detailed description of the results of the later morbidity data set. It is not inconsistent with the significant results cited by Robinette et al. but it does show a wider range of significant adverse health effects.

In the later VA compensation data Robinette et al. found significantly increase in sickness for Musculoskeletal system and other organs, including:

Loss of part extremities, osteomyelitis and neoplasms of bone or muscle ($p<0.001$);
Organs of special sense which includes eye cataracts ($p<0.05$);
Respiratory system, excluding pulmonary tuberculosis ($p<0.01$);
Cardiovascular system ($p<0.001$); and
Mental disorders, including psychoses, psychoneurotic disorders and so-called "psychophysiologic disorders" ($p<0.05$).

The Table 11 shows all of the diagnosis groups detailed in Robinette et al. Table 12. For VA compensation claims up to December 1976. Again the vast majority of symptoms (apart from Nerves, and Genitourinary) are marginally significant to very significantly greater for the higher exposed FT+AT group compared to the lower exposed ET group. Except for "Nerves" all symptoms are elevated and some, as also identified by Robinette et al., are significantly and highly significantly elevated.

Table 11: Number of men receiving VA compensation and pension, December 1976 and rates per 1000 men per year by diagnosis and exposure class, and Risk Ratio (FT+AT)/ET, Robinette et al. Table 12.

	ET		FT+AT		Risk Ratio		Significance
	No.	Rate	No.	Rate	RR	95% CI	p-value
Diagnosis:							
Musculoskeletal	115	8.8	119	16.9	1.93	1.49-2.49	<0.001
Organs of special sense	49	3.7	42	6.0	1.62	1.07-2.45	0.010
Systematic conditions	3	0.2	5	0.7	3.50	0.84-14.65	0.080
Respiratory	55	4.2	51	7.3	1.74	1.19-2.55	0.001
Cardiovascular	43	3.3	47	6.7	2.03	1.34-3.07	<0.001
Digestive	74	5.7	55	7.8	1.37	0.97-1.94	0.02
Genitourinary	31	2.4	10	2.7	1.13	0.55-2.30	0.32
Skin	83	6.3	58	8.2	1.30	0.93-1.82	0.052
Endocrine	15	1.1	11	1.6	1.45	0.67-3.16	0.86
Neurological	21	1.6	16	2.3	1.44	0.75-2.76	0.29
Nerves	15	1.1	3	0.4	0.36	0.10-1.24	0.14
Mental Conditions	51	3.9	46	6.5	1.67	1.12-2.49	0.003

Discussion of Results:

This project was conducted with the objective of determining whether radar exposure to service personnel during the Korean War produced health hazards. Despite significant time delay dilution, the data still contains significant dose response relationships and may significant to extremely significant increases in mortality and illness. This data shows that radar exposure is (through extremely significant increases and significant dose-response relationships) causally related increased mortality and illness, including cancer and diseases in many body organs. Radar exposes the whole body and causes sickness across the whole body.

Hence the WHO, ICNIRP and authors conclusions are wrong.

Global Leukaemia dose response for RF/MW exposure:

Leukaemia is frequently significantly raised in RF/MW exposed populations. Table 12 summarizes several studies that are ranked in mean exposure order. Military, occupational and residential studies shows a global dose response relationship for increased adult leukaemia and RF/MW exposure with a global dose-response relationship and a threshold close to zero.

The Polish Military Study, Szmigielski (1996), contrary to the comments in ICNIRP (1998), is a very large and well defined study, carefully designed and executed by Dr Stanislaw Szmigielski and his team at the Polish Center for Radiobiology and Radiation Safety at the Military Institute of Hygiene and Epidemiology. To be classified as exposed the exposure must be recorded and assessed. The extremely significant leukaemia increases in $p<0.001$, are adequate to show a causal relationship. This is confirmed by the many other studies showing that RF/MW radiation is significantly associated with leukaemia. The study also shows elevated to significantly elevated cancer across many body organs

When actual residential exposures are considered, dose-responses for residential cancer are also shown by Selvin et al. (1992), Hocking et al. (1996), Dolk et al. (1997 a,b) and Michelozzi et al. (1998). These strongly confirm a causal relationship with adult and childhood leukaemia because the radial RF/MW exposure and cancer rates match and produce highly significant dose-response relationships. The Sutton Coldfield Study also supports the evidence that RF exposure causes cancer in many body organs.

Table 12: A summary of epidemiological studies involving adult leukaemia mortality or incidence, ranked by probable RF/MW exposure category.

Study	Reference	Exposure Category	Leukaemia Type	Risk Ratio	95% Confidence Interval
Polish Military (Mortality)	Szmigielski (1996)	High	ALL	5.75	1.22-18.16
			CML	13.90	6.72-22.12
			CLL	3.68	1.45-5.18
			AML	8.62	3.54-13.67
			All Leuk.	6.31	3.12-14.32
Korean War Radar Exposure	Robinette et al. (1980) (Mortality)	High/Low AT/(RD+RM)	Leuk/Lymp	2.96	1.39-6.32
Radio and TV Repairmen	Milham (1985)	Moderate	Acute Leuk.	3.44	
			Leuk.	1.76	
Amateur Radio (Mortality)	Milham (1988)	Moderate	AML	1.79	1.03-2.85
UK Sutton Coldfield <=2km	Dolk et al. (1997a)	Moderate	Leuk	1.83	1.22-2.74
North Sydney TV/FM towers (Mortality)	Hocking et al.(1996)	Low	All Leuk.	1.17	0.96-1.43
			ALL+CLL	1.39	1.00-1.92
			AML+CML	1.01	0.82-1.24
			Other Leuk	1.57	1.01-2.46
UK TV/FM (Incidence)	Dolk et al. (1997b)	Low	Adult Leuk.	1.03	1.00-1.07

Note: ALL : Acute Lymphatic Leukemia; CLL: Chronic Lymphatic Leukaemia; AML Acute Myeloid Leukaemia; CML: Chronic Myeloid Leukaemia; and All Leuk.: All Adult Leukaemia.

Residential Cancer Studies:

Mean Personal Exposures:

Personal exposures will be somewhat less than the direct peak exposure at a given location. McKenzie, Yin and Morrell (1998): On the Roof: 3.0 $\mu W/cm^2$; At Street Level:

0.066 μW/cm^2; and In the House: 0.017 μW/cm^2. This gives a reduction factor of 1: 45 : 176. This is reduced further to 1 : 20 : 50 for the following estimate.

By estimating the time spent inside and outside, at home and away, a mean residential exposure factor is obtained. Of 168 hours/week the Ratio is estimated as: exposed:outside:away:inside is 6: 20: 12: 130 . This gives a residential exposure factor (REF) of 0.061. This is rounded up to REF = 0.075.

Exposure Dilution:

All observed Odds and Rate Ratios will be significantly reduced because of a number of dilution factors, including:

- EMR is Ubiquitous
- Reference Group is also exposed
- Randomization over intervening decades;
- Migration around Towers.

The migration factor results from exposed people moving away and unexposed people moving into the study area.

Horizontal antenna patterns:

The vital feature of residential studies is the complex horizontal and radial RF exposure patterns. In multiple studies these patterns match the cancer patterns, confirming the causal relationship, even at the very low residential exposure levels.

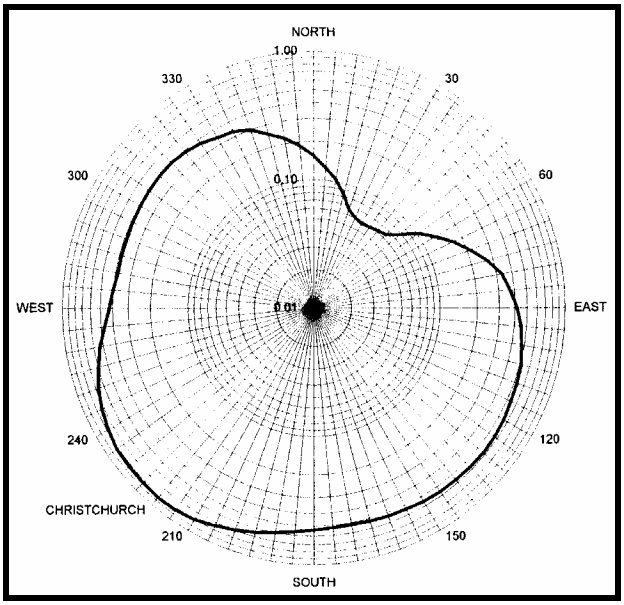

Figure 29: Horizontal antenna pattern for an 8-element dipole array for a 98 MHz FM transmission.

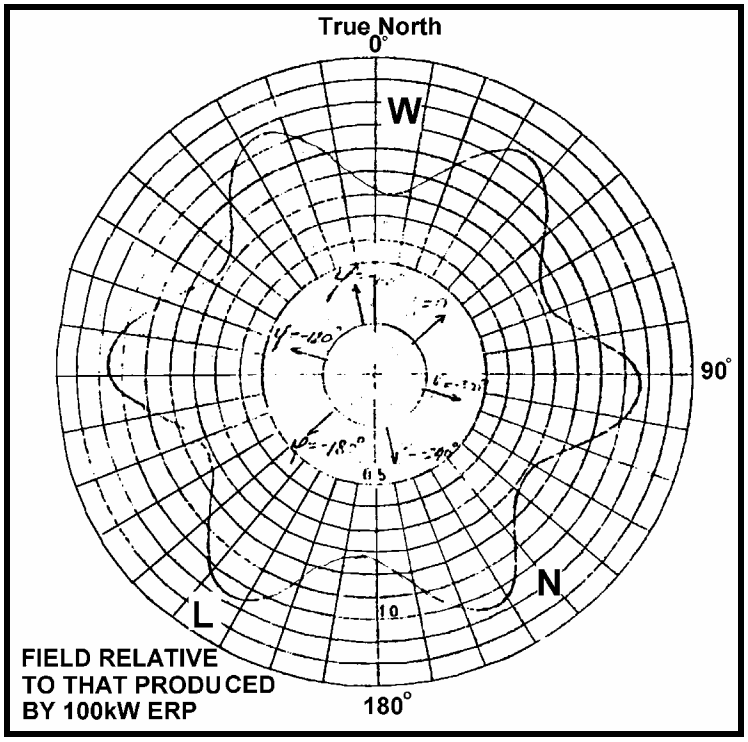

Figure 30 : Horizontal VHF antenna pattern from a North Sydney transmitter.

McKenzie at al. (1998) criticize Hocking et al. (1996). They provide estimated exposures for the centroid of each municipality and childhood leukaemia incidence rates for each municipality. They are unaware of the horizontal radiation patterns that bias the signals towards the major population areas to the SW of the towers, Figure 30. This puts the highest signal over Lane Cove (L), a middle strength signal over North Sydney (N) and a weaker than average signal over Willoughby. By adjusting the McKenzie estimated by the horizontal pattern (in dB) the exposure levels are L = 1.46, N = 0.29 and W = 0.27μW/cm^2. The cancer rates are L = 16.7, N = 9.6 and W = 6.1 per 100,000, a dose-response.

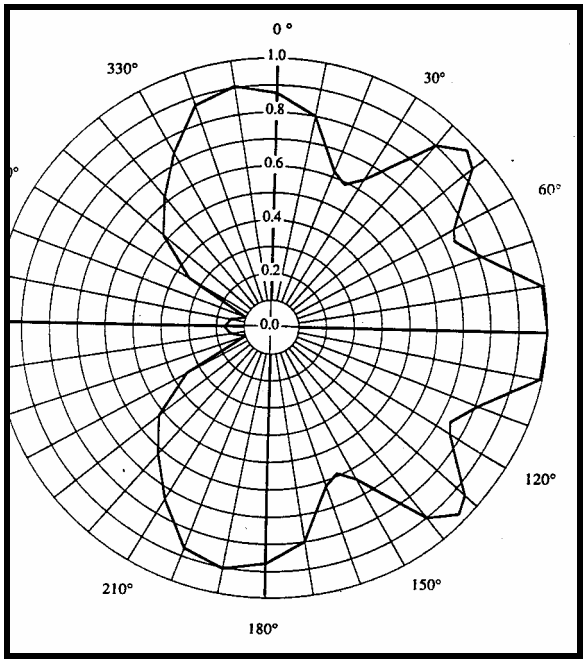

Figure 31: Horizontal antenna radiation patterns showing the relative filed strength for, (a) UHF Digital TV (linear scale) from the Sutra Tower.

Vertical Antenna Patterns:

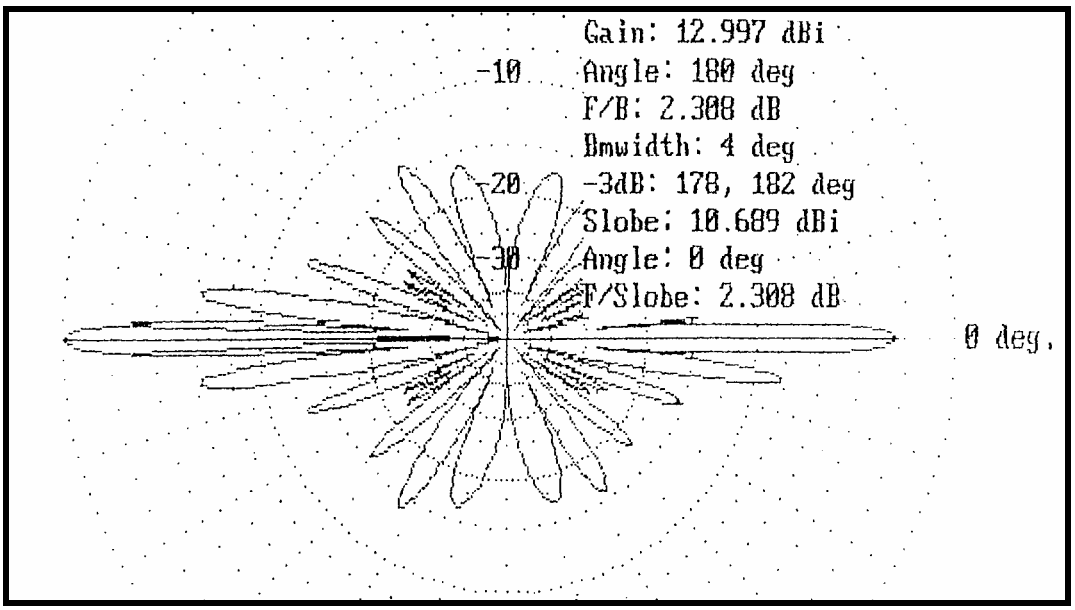

Figure 32: A typical vertical antenna pattern for a 4-element dipole array at about 98 MHz.(VHF), Units in dB. This produces peaks close to the tower.

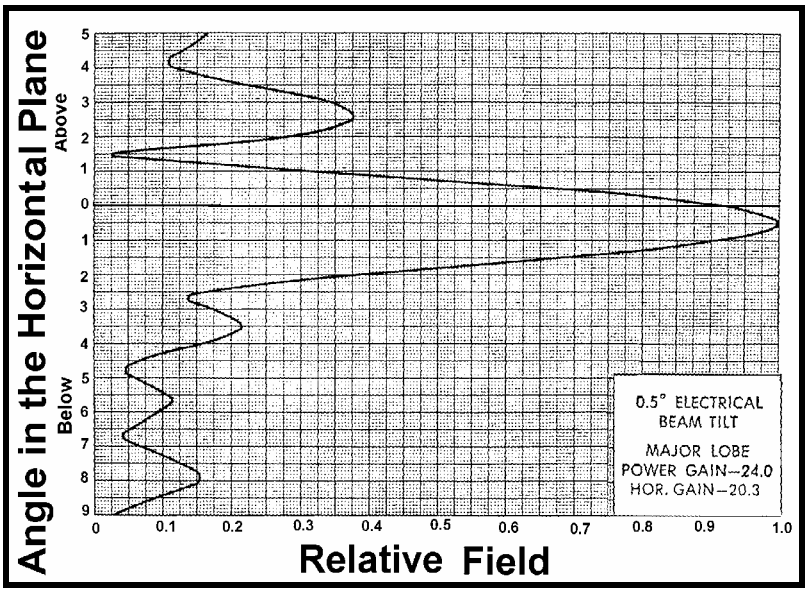

Figure 33: A UHF relative field factor (RFF) for the vertical antenna pattern from Hammett and Edison (1998).

Radial Exposure Patterns:

UHF - Type A pattern, Low, elevated undulating, low to 10 km.

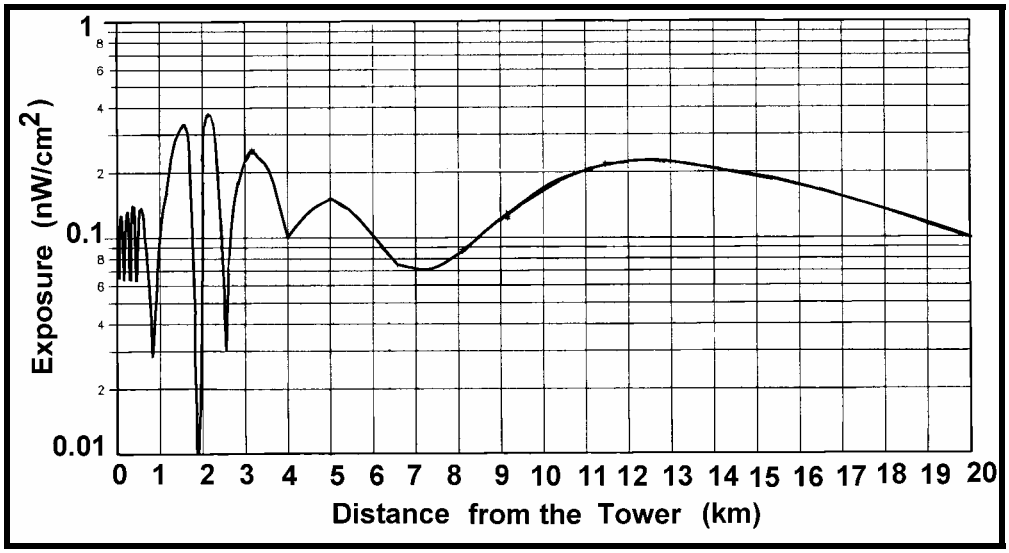

Figure 34: Ground level exposure for a typical UHF TV broadcast signal, from an antenna pattern from Hammett and Edison (1997), for a 20 MW EIRP transmitter at 450m AGL, for a flat surface.

VHF - Type B pattern, high near the tower and declining in an undulating fashion with distance.

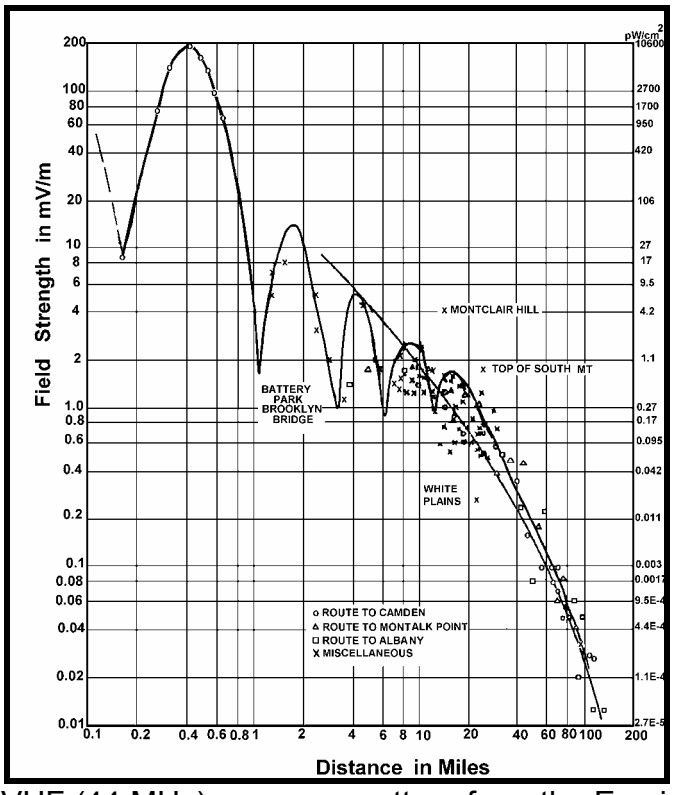

Figure 35: A typical VHF (44 MHz) exposure pattern from the Empire State Building, New York, Jones (1933).

Sutro Tower Study:

Selvin et al. (1992) studied the spatial distribution of 4 childhood cancers in relation to the Sutra Tower in San Francisco. When measured and practical radial exposure patterns are

compared with the radial cancer rates a highly significant dose response relationship results, Figure 36.

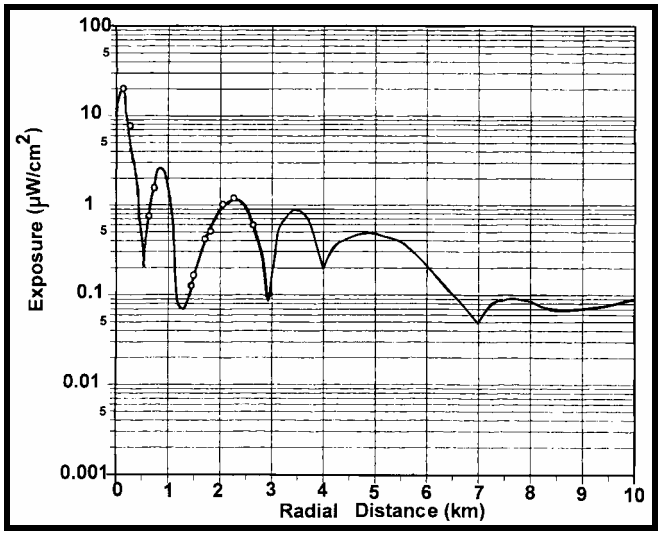

Figure 36: The measured and estimated power density (exposure in $\mu W/cm^2$) with distance from the Sutra Tower. Circles show measurements. The line follows measurement points and the radial pattern of a typical UHF transmission beyond 3 km. From Hammett and Edison (1997) and readings taken by the author in 1999.

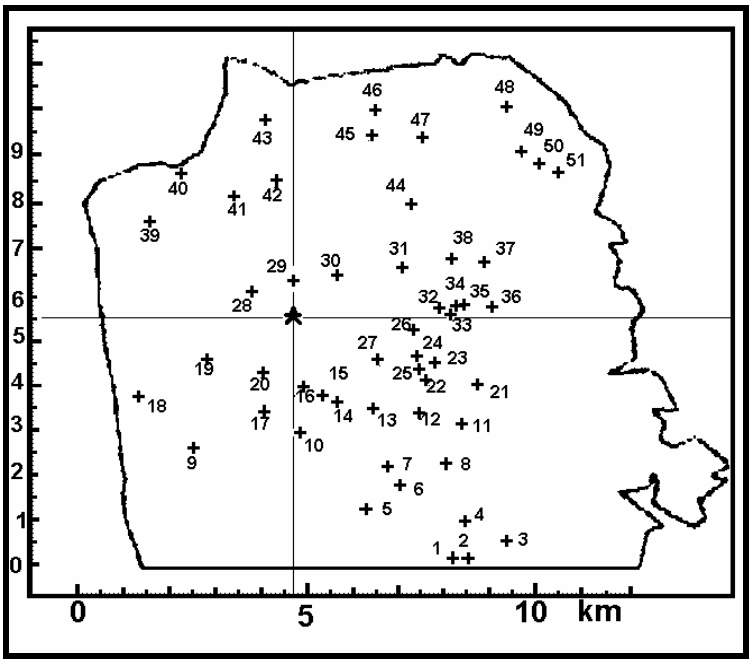

Figure 37: Spatial map of white childhood (<21 years) leukaemia for San Francisco, 1973-88, from Selvin et al. (1992).

Because of the complex nature of residential radial broadcast tower exposure patterns, Figure 36, the chance of confounding effects are extremely small. Thus this indicates a causal relationships Plotting the radial residential mean exposure and the "All Cancer" Risk Ratio gives the pattern in Figure 38. The match shows that no other factor can explain this result than the RF exposure from the Sutra Tower.

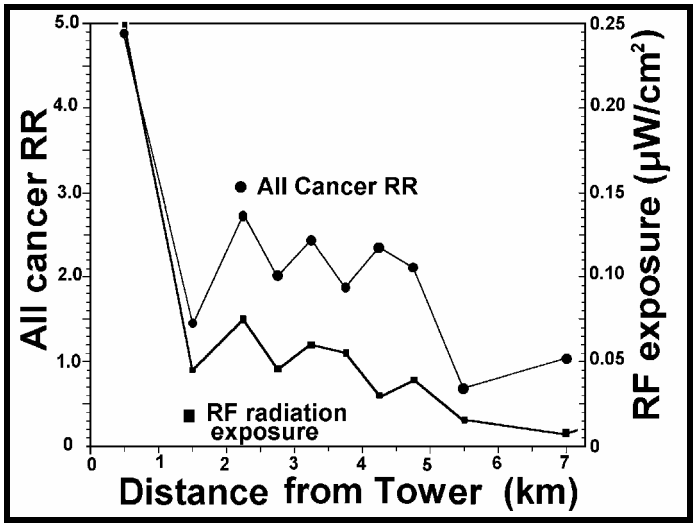

Figure 38: The radial All Cancer Risk Ratio and the mean residential RF exposure (times 20 to fit on the scale).

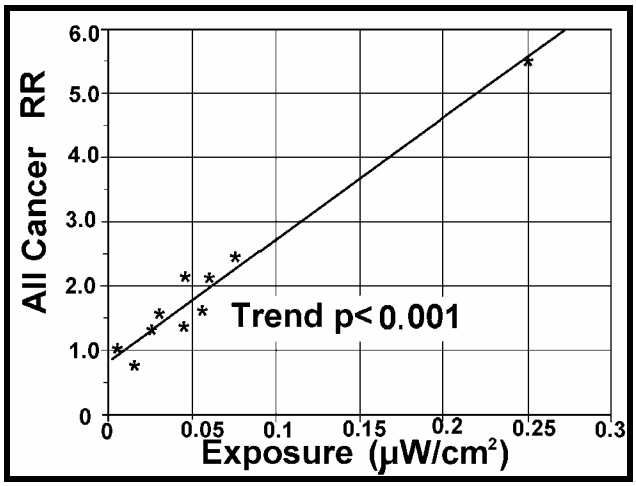

Figure 39: All Cancer Risk Ratio for Childhood Cancer as a function of estimated radial group mean personal exposure to RF/MW radiation from the Sutra Tower, San Francisco, using the spatial childhood cancer data presented in Selvin et al. (1992). The dose-response relationship is extremely significant (p<0.001).

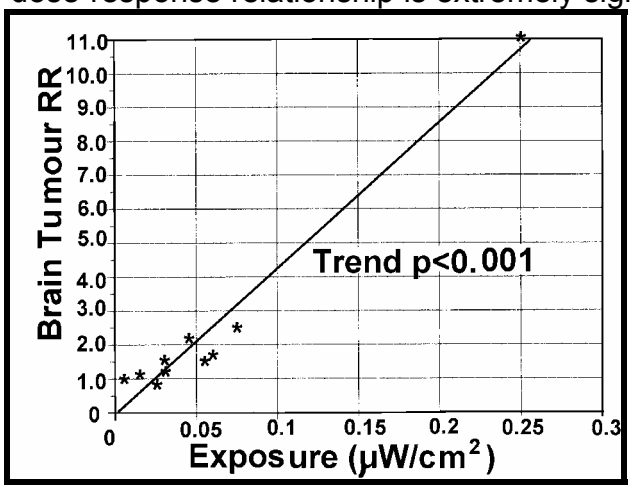

Figure 40: Brain Tumour Risk Ratio as a function of estimated radial group mean personal exposure to RF/MW radiation from the Sutra Tower, San Francisco, using the spatial childhood cancer data presented in Selvin et al. (1992). The linear dose-response relationship is extremely significant (p<0.001).

Within the data uncertainty, the dose-response threshold is zero. Hence RF/MW is causally associated with adult and childhood cancer, including leukaemia with a dose-response relationship with a zero exposure threshold.

United Kingdom Regional TV Tower Study: Dolk et al. (1997)

The Study Context:

Dr Helen Dolk and her colleagues responded to concerns about a cluster of seven cases of leukaemia and lymphoma who were patients of a Birmingham GP, Dr Mark Payne, and who lived near the Sutton Coldfield Transmitter. They obtained data from the cancer registry and found a high incidence of adult leukaemia near the tower, which declined with distance. They assumed that this was a dose-response relationship that was following an inverse square law for exposure decline with distance from the transmitter. Before they published this result they decided to extend the study to 20 other regional TV towers throughout the United Kingdom.

At these individual sites, and for all the 20 sites combined, the adult leukaemia rate was found to be low near the tower, rose to form a broad variable peak between about 1 km and 5 km, and then declined with distance. Over all distance It didn't follow an inverse square law and therefore it failed to confirm the result found at Sutton Coldfield, Figure 41.

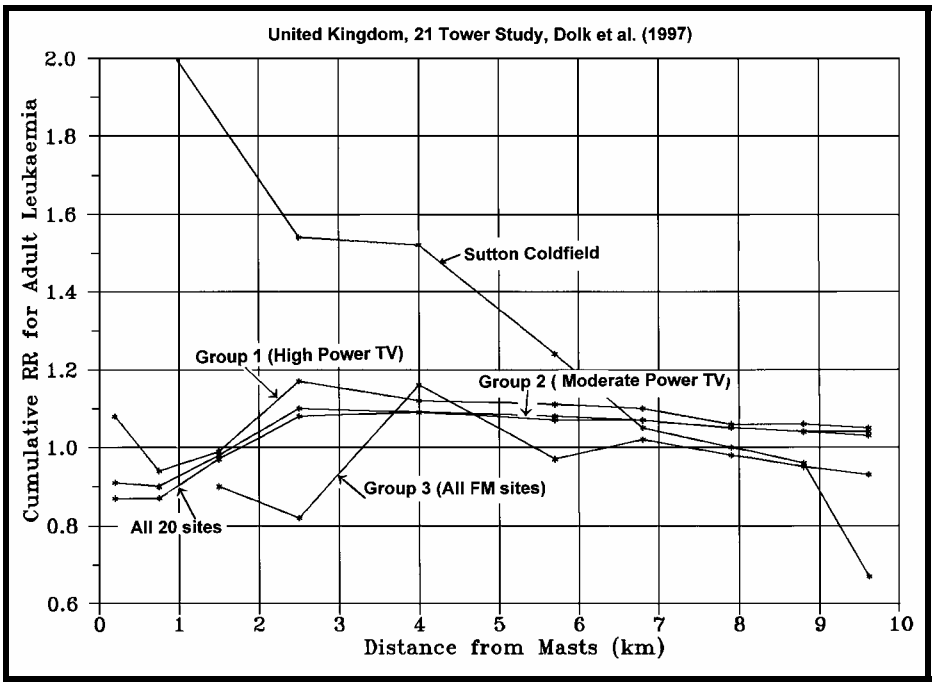

Figure 41: Radial adult leukaemia patterns for the 21 site UK study, Dolk et al.

Thus Dolk et al. (1997b) concludes that the follow-up study "at most gives very weak support to the Sutton Coldfield findings." ICNIRP accepts this conclusion and states that the results of these U.K. studies "are inconclusive".

There are two types of radial transmission signals and two types of radial cancer patterns:

Type A : UHF signals that are low near the tower, rise to a broad peak between 2 and 6 km and then decline with distance, Figure 33.

Type B: VHF signals have a peak within 1 km and decline with distance in an undulating fashion, Figure 34.

For a high cancer rate to be detectable near a tower three factors are necessary:

1. There must be a large population. This requires a high population density because there is only a small area within 1 km radius of the tower and a high proportion of this is likely to be the open field in which the tower itself is sited.

2. There needs to be a high radiation exposure for the radiation to be able to elevate the cancer rate. This occurs for the lower frequency, VHF, FM signals, Figures 34.

3. The cancer type needs to be RF-radiation sensitive to assist in raising the cancer incidence above the background level. Leukaemia and Lymphoma are very RF-sensitive cancers, Szmigielski (1996), Milham (1985, 1988), Hocking et al. (1996).

These factors completely explain these results. Sutton Coldfield is the only tower that has these three factors. All other towers lack at least one factor and therefore cannot show a high cancer rate near the tower. In fact they all follow a Type A pattern which is a dose response relationship of cancer rate as a function of mean exposure. This for all radial cancers outlined in the Tables they follow a dose response relationship appropriate to their radiation patterns.

The data in Dolk et al. is internally consistent, shows elevated childhood leukaemia and brain tumor, and a set of dose-response relationships which are likely to be highly significant, if related to realistic radial RF patterns, for cancer at a wide range of body sites including All Cancer, Leukaemia, Non-Hodgkin's Lymphoma, Brain Cancer, Bladder Cancer, Prostate Cancer, Skin Melanoma, Male and Female Breast Cancer and Colorectal Cancer. This is also consistent with Robinette et al. (1980), Szmigielski (1996) and Milham (1985, 1988).

Sleep Disturbance near a Shortwave Radio Tower, Schwarzenburg, Switzerland:

The Schwarzenburg Study, Alpeter et al. (1995) and Abelin (1999) showed a causal relationship of sleep disturbance with exposure to a short wave radio signal. The effect is assessed as causal because of the significant dose response relationship, the variation of sleep disturbance in two experiments, one involving changing the beams and one turning the transmitter off, and the identification of significant melatonin reduction. Professor Abelin told seminars in Christchurch that they had measured a significant increase in melatonin after the tower transmission was turned off permanently compared to the levels while it was on. Measurements of salival melatonin in two herds of 5 cows revealed a significant rise in melatonin in the exposed cows when the tower was turned off for three days, Figure 42.

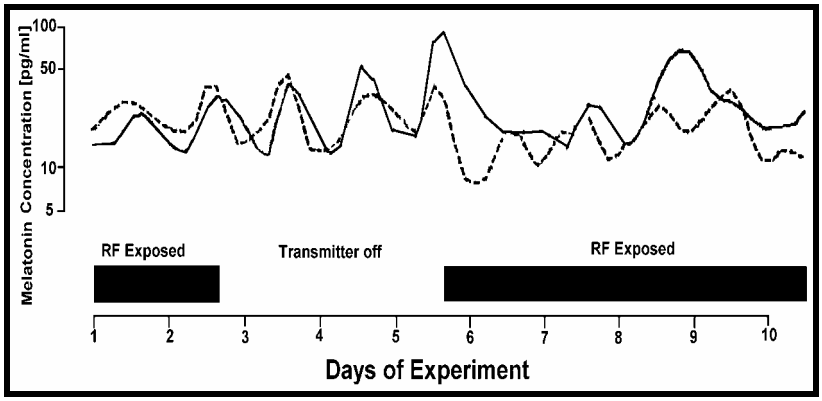

Figure 42: Salival melatonin from two herds of 5 cows, one exposed at 500 m, 0.095μW/cm^2, (solid line) and one "unexposed" at 4000 m, 0.00022μW/cm^2, (dashed line).

On average the exposed herd had lower melatonin, but not significantly so because of the very small sample size. The same difference with about twice as many cows would have been significant.

Figure 42 also reveals that when the tower was turned on the "unexposed" herd showed a drop in melatonin. Under normal tower operation the exposed cows had a delay in their nocturnal peak by 2 to 3 hours.

When the tower was turned off the sleep quality improved significantly for the three groups being monitored at that time. Figure 43 shows the results for the highest and lowest exposed groups, Group A and Group C.

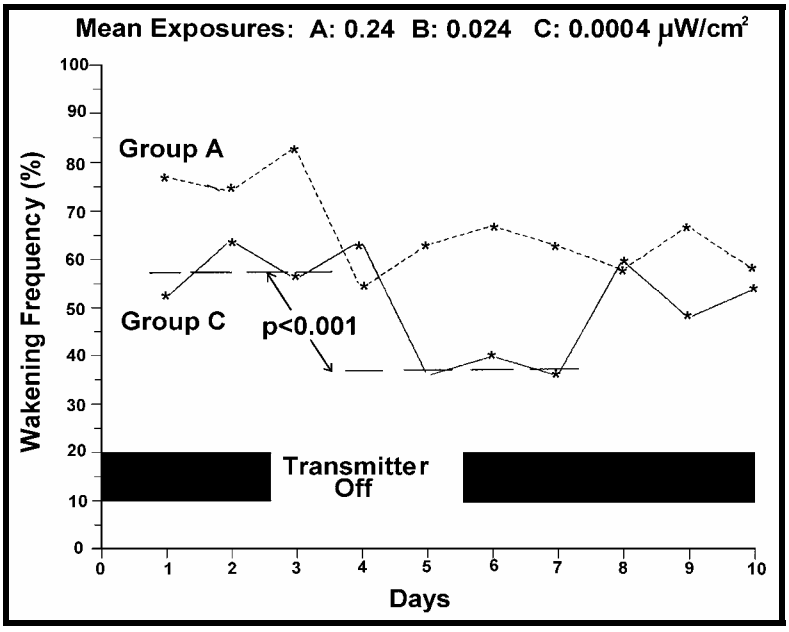

Figure 43: Sleep disturbance in people exposed to a short-wave radio stations which was turned off for three days, Altpeter et al. (1995), showing the highest exposed Group A, and lowest exposed Group C.

Both Groups show a delayed improvement in sleep of one to two days. The reduced wakening averaged over days 4 to 6 compared with days 1 to 3 are highly significantly

reduced, p<0.001. Thus the lowest exposed group, 0.0004μW/cm^2 also shows a significant effect of the RF exposure on sleep disturbance.

Thus turning the tower off revealed significant rises in bovine melatonin and human sleep quality. Human melatonin increased significantly when the tower was turned off permanently. Groups B, R and C are all exposed to a mean RF signal of less than 0.1μW/cm^2 and they experienced highly significant sleep disturbance and reduced melatonin.

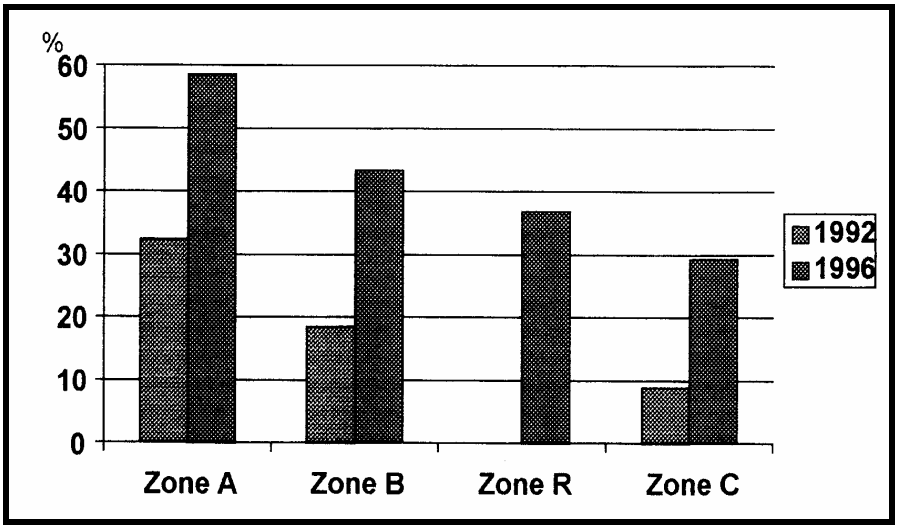

Figure 44: Adult Sleep Disturbance with RF exposure at Schwarzenburg, Switzerland, Abelin (1999).

Sleep disruption occurs in a dose-response manner with a threshold below 0.1nW/cm^2, i.e. very close to zero, Figure 45.

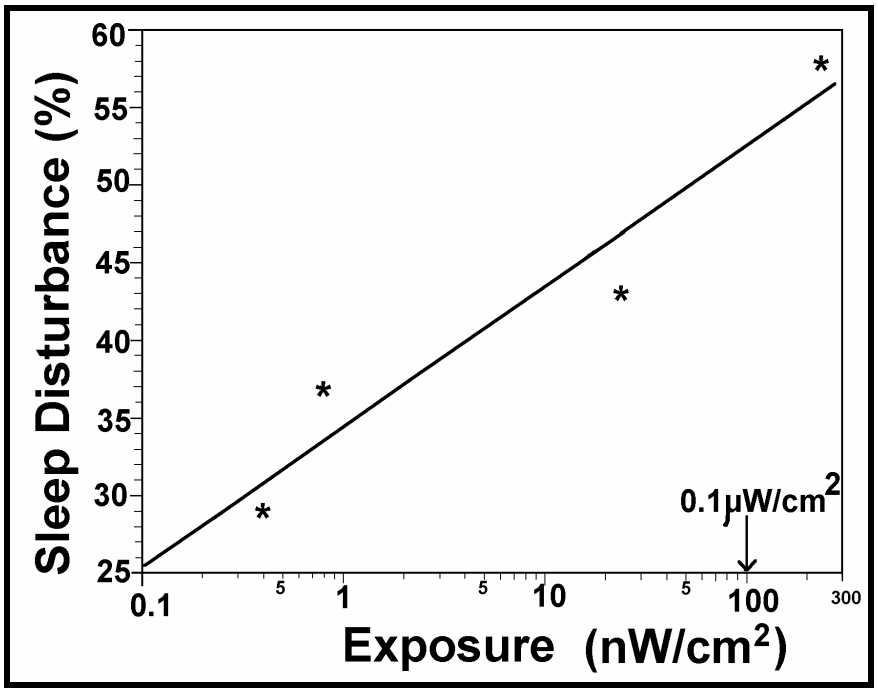

Figure 45: Dose-response relationship for Sleep Disturbance at Schwarzenburg with exposure in nW/cm2. Note: 1nW/cm^2= 0.001μW/cm^2

Since sleep disturbance, Mann and Roschkle (1995), and melatonin reduction, Burch et al. (1997), has been observed with cell phone exposure. Hence these observations also apply to cell phones and cell sites.

Broadcast Tower Conclusions:

The Swiss researchers in the Schwarzenburg Study concluded that there was a causal relationship with sleep disruption and exposure to RF radiation. This shows the exquisite sensitivity of the brain to RF radiation, reduction in a vital neurohormone, melatonin, which is related to sleep quality, chronic fatigue and cancer. The Schwarzenburg study also identified a suite of symptoms that they referred to as Chronic Fatigue. In the U.K., Australia, San Francisco, Hawaii and Italy residential studies above show significant increases in adult and childhood leukaemia and multiple significant dose response relationships for a range of cancers, especially leukaemia and brain tumour and all cancer at residential exposure levels.

This forms a coherent, consistent, integrated set of studies showing a causal relationship between sleep disturbance, chronic fatigue and cancer in association with extremely low mean RF exposure levels experiences in residential situations in the vicinity of radio and TV transmission towers.

Summary of ICNIRP's assessment: Critique

The cancer assessment, ICNIRP (1998) p 504, references one review (UNEP/WHO/IRPA 1993), WHO (1993), and 13 papers covering 11 studies. The WHO (1993) review, is limited by citing only 6 epidemiological studies and, by not reviewing the actual results, contains errors, which are propagated through to the ICNIRP assessment.

In ICNIRP (1998), only 13 papers are cited directly:

Barron and Baraff (1958): The study group is too small (226) and the follow up period (4-13 years from first exposure) is too short to detect cancer. Cancer is not one of the paper's studies chosen outcomes. It is grossly dishonest and misleading to include this paper in a cancer assessment and to cite it as showing that there are no cancer risks from exposure to radar.

Robinette et al. (1980): Is widely claimed to show no effects when its data does show many significant adverse human health effects, including a significant dose-response relationship for all mortality, cancer, leukaemia and respiratory cancer.

Lilienfeld et al. (1978): Is widely claimed to show no effects when its data does show significant adverse human health effects, including neurological, cardiac and cancer effects and includes a significant dose-response relationship for rates of sickness as a function of years in Moscow.

Selvin et al. (1992): Is widely claimed to show no effects when it was aiming to develop an epidemiological method relating to spatial clustering. Its data does show significant adverse human health effects, including significant dose-response relationships when radial cancer rates are related to radial exposure measurements.

Beall et al. (1996): Is quoted by ICNIRP as failing to show significant increases in nervous system tumours, when it does, and includes a significant dose-response relationship between years of exposure and rates of brain tumor for computer programmers.

ICNIRP (1998) claims that this study showed no significant increases in nervous system tumours. This is factually wrong. The overall results of Beall et al. (1996), as presented in their abstract is: There was elevated ORs:

"For 10 or more years of employment in engineering/ technical jobs (OR = 1.7, 95% CI: 1.0-3.0) or in programming jobs (OR = 2.8, 95% CI: 1.1-7.0). The OR for glioma for all subjects who had accrued 5 years of programming work 10 years before the case's death was 3.9 (95% CI: 1.2-12.4)."

For engineers/technicians the dose-response has p=0.07. For computer programmers, those most using computers, the dose-response is significant, p = 0.04.

6. **Grayson (1996)** Is quoted by ICNIRP as failing to show significant increases in nervous system tumours, when it does show a significant increase in brain tumor for RF/MW exposed personnel.

   Grayson carried out a job title-time-exposure matrix utilising potential intensity scores for both ELF and RF/MW EMR exposures. Data on ionizing radiation exposure was also available.

   "Although the present study has its limitations, particularly in exposure estimation, it does suggest that there is a small association between potential EMF exposures and brain tumor risk among Air Force members, especially for personnel potentially exposed to Radiofrequency/microwave EMFs."

   The results for the three types of radiation exposure, after adjustment for: Age-race-senior military rank, were:

Ionizing Radiation	OR = 0.58	95%CI: 0.22-1.52
ELF Radiation	OR = 1.28	95%CI: 0.95-1.74
RF/MW radiation	OR = 1.39	95%CI: 1.01-1.90

   The RF/MW exposure gives a significant 39% increase in brain tumor.

7. **Rothman et al. (1996a):** ICNIRP acknowledges that it is still too early to observe an effect of cancer incidence and mortality from mobile telephone use as yet.

8. **Rothman et al (1997b)** ICNIRP acknowledges that it is still too early to observe an effect of cancer incidence and mortality from mobile telephone use as yet. However the study does show a significant increase in the mortality of old whole body exposing mobile phone users compared with smaller, hand held, head exposing portable phone users, RR = 1.38, 95%CI: 1.07-1.79, p=0.013.

9. **Szmigielski et al. (1988):** finds significant increases in cancer across the body, especially leukaemia incidence and mortality among Polish Military personnel

exposed to radio and radar. ICNIRP says is difficult to interpret because neither the size of the population nor the exposure levels are clearly stated. In fact the Polish Military microwave exposure regime is presented and the group is described by the authors as “large and well controlled”.

10. **Szmigielski (1996):** ICNIRP acknowledges that Szmigielski found significant increases in leukaemia but criticizes the exposure assessment and the description of the population. Again, the overall group exposure regime is well described, but as in all large population studies, individual exposures are not monitored but group exposures can be well classified. The Leukaemia results are so extremely significant as to be considered as causal.

11. **Hocking et al. (1996),** (**12**.) **Dolk et al. (1997a)** and (**13**.) **Dolk et al. (1997b)** are acknowledged as “suggesting a local increase in leukaemia incidence” in populations living in the vicinity of TV/FM transmission towers, but ICNIRP calls the results “Inconclusive”. The UK studies of Dolk et al., show a causal relationship with radial distance and Hocking strongly supports this with a dose-response increase.

ICNIRP's overall cancer assessment conclusion that: "*Overall, the results of the small number of epidemiological studies published provide only limited information on cancer risk*."

This conclusion is mistakenly based on flawed previous assessments, WHO (1993), inappropriate inclusion of (1) and (7), failure to review the data on effects (2, 3, and 4), incorrect claims of no significant effects when such effects are reported (5 and 6), failure to analyse the data in (8), inappropriate dismissal of significant well conducted studies (9 and 10) and inappropriate devaluing of residential studies (11, 12 and 13).

All of the six appropriately included studies that ICNIRP claims to show no effects, in fact do show significant effects and five show significant to highly significant dose-response relationships. The five studies that ICNIRP agreed do show effects, but seeks to dismiss them, are all wrongfully dismissed. They also contain highly significant effects and significant dose-response relationships for residential RF/MW exposures.

A systematic and independent analysis of the data in these papers reveals a consistent and significant increase in cancer with many dose response realtionships in this set of studies. Also, many other studies exist which add considerable weight to the conclusion that there is a causal relationship between RF/MW and brain cancer, leukaemia and cancer across many organs in the body. The no-effects threshold is zero exposure.

Cellphone radiation Mimics RF/MW Effects:

Because cellphone radiation is also genotoxic, causes brain tumours, cardiac and neurological effects, including sleep disturbance and melatonin reduction, all of these effects will be associated with cell sites.

The Issue:

Thousands of people are using cell phones for hours each day. They are exposing a very sensitive organ, their brain, to higher mean intensities than military personnel are exposed to when repairing radar. The military personnel show significant increases in cancer and a

wide range of illnesses. Even at the very low mean levels that people experience living within 10 km of radio and TV towers, significant increases in cancer has been observed.

Analogue cell phones emit an analogue modulated RF/MW signal similar to an FM radio or TV signal. The digital cell phones radiate a pulse RF/MW signal similar to a radar. Biological and epidemiological effects from EMR exposure across the spectrum show the same or similar effects.

Many people continue to drive while talking on their cell phones. Attention deficit and neurological effects on the user's brain make accidents much more likely.

Very young children and teenagers are becoming regular to heavy users of cell phones while their brains and bodies are in a much more vulnerable state than elderly people. With cancer and neurodegenerative disease latencies of decades, the possible adverse effects will take some time to become evident. By which time it will be too late for thousands of people.

There is growing concern about cell phone interference with cardiac pacemakers. If cell phone signals can interfere with an electronic pacemaker, then it is likely to also interfere with human hearts that are arrhythmically unstable.

Effects shown for electromagnetic radiation, especially radio and radar signals:

Such signals have been shown to:

- Alter brain activity, including EEG and reaction times, memory loss, headaches, fatigue and concentration problems, dizziness (the Microwave Syndrome), Gordon (1966), Deroche (1971), Moscovici et al. (1974), Lilienfeld et al. (1978), Shandala et al. (1979), Forman et al. (1982), Frey (1998).

- Impair sleep and learning, Altpeter et al. (1995), Kolodynski and Kolodynska (1996)

- Increase permeability of the blood brain barrier (a mechanism for headache), Frey et al. (1975), Alberts (1977, 1978) and Oscar and Hawkins (1977).

- Alter GABA, Kolomytkin et al. (1994).

- Increase neurodegenerative disease including Alzheimer's Disease, Sobel et al. (1995, 1996), Savitz et al. (1998a,b)

- Highly significant Increased permeability of the blood brain barrier for 915 MHz radiation at SAR =0.016-0.1 (p=0.015) and SAR = 0.1-0.4 (p=0.002); Salford et al. (1994).

- Alter blood pressure and heart rhythm (heart rate variability) and Heart Disease, Forman et al. (1986), Hamburger, Logue and Silverman (1983), Bortkiewicz et al. (1995, 1996, 1997) and Szmigielski at al (1998), Savitz et al. (1999)

- Increase the Suicide Risk, Baris and Armstrong (1990), Perry et al. (1991), Van Wijngaarden et al. (2000).

- Impair the immune system Quan et al. (1992), Dmoch and Moszczynski (1998), Bruvere et al. (1998)

- Reduce sperm counts, Weyandt et al. (1996)

- Increase miscarriage and congenital abnormalities, Kallen et al. (1982), Larsen et al. (1991), Ouellet-Hellstrom and Stewart (1993).

- Reduce melatonin and alter calcium ions, Abelin (1999), Burch et al. (1997, 1999) Bawin and Adey (1976), Blackman et al. (1988, 1989, 1990).

- Enhances heat shock proteins at extremely low exposure levels in a highly reproducible manner showing that they are not stimulated by heat but in reaction to a 'toxic' protein reaction, Daniells et al. (1998), and down to 0.001W/kg (0.34μW/cm^2) using 750MHz microwaves, de Pomerai (2000).

- Break DNA strands, damage chromosomes, alter gene transcription activity, and neoplastically transform cells. Lai and Singh (1995, 1996, 1997), Garaj-Vrhovac et al. (1990, 1991, 1992, 1993, 1999), Vijayalaxmi et al. (1997), Phillips et al. (1992, 1993), and Balcer-Kubiczek and Harrison (1991).

- Enhances cell death in a dose response manner for signal intensity and exposure time, Garaj-Vrhovac et al. (1992).

- Enhances cell proliferation in a dose-response manner for exposure time, Mattei et al. (1999).

- Enhances Ornithine Decarboxylase (ODC) activity, a measure of cell proliferation rate, Byus et al. (1988), Litovitz et al. (1997).

- Enhances free radicals, Phelan et al. (1992)

- Increase the incidence of many types of cancer, including leukaemia, brain tumor, testicular cancer, genitourinary and breast cancer, Robinette et al. (1980), Milham (1985, 1988), Szmigielski (1996), Hocking et al. (1996), Dolk et al. (1997 a, b), Beall et al. (1996), Grayson (1996), Thomas et al. (1987), Lilienfeld et al. (1978), Zaret (1989), Davis and Mostofl (1993), Hayes et al. (1990), Tynes et al. (1996), Cantor et al. (1995).

These biological and health effects are consistent with the biological understanding that brains, hearts and cells are sensitive to electromagnetic signals because they use electromagnetic signals for their regulation, control and natural processes, including those processes monitored by the EEG and ECG. There is overwhelming evidence that EMR is genotoxic, alters cellular ions, neurotransmitters and neurohormones, and interferes with brain and heart signals, and increases cancer.

Cell Phone Radiation Research:

For years the cell phone companies and government authorities have assured us that cell phone are perfectly safe. They state that the particular set of radiation parameter

associated with cell phones are not the same as any other radio signal and therefore earlier research does not apply. They also mount biased review teams who falsely dismiss any results that indicate adverse biological and health effects and the flawed pre-assumption that the only possible effect is tissue heating. There is a very large body of scientific research that challenges this view. Now we have published research, primarily funded by governments and industry that shows that cell phone radiation causes the following effects:

- Alters brain activity including EEG, Von Klitzing (1995), Mann and Roschkle (1996), Krause et al. (2000).

- Disturbs sleep, Mann and Roschkle (1996), Bordely et al. (1999)

- Alters human reaction times, Preece et al. (1999), Induced potentials, Eulitz et al. (1998), slow brain potentials, Freude et al. (1998), Response and speed of switching attention (need for car driving) significantly worse, Hladky et al. (1999). Altered reaction times and working memory function (positive), Koivisto et al. (2000), Krause et al. (2000).

- Weakens the blood brain barrier ($p<0.0001$): Persson, B.R.R., Salford, L.G. and Brun, A., 1997.

- A Fifteen minute exposure, increased auditory brainstem response and hearing deficiency in 2 kHz to 10 kHz range, Kellenyi et al. (1999).

- While driving, with 50 minutes per month with a cell phone, a highly significant 5.6-fold increase in accident risk, Violanti et al. (1996); a 2-fold increase in fatal accidents with cell phone in car, Violanti et al. (1998); impairs cognitive load and detection thresholds, Lamble et al. (1999). In a large Canadian study Redelmeier and Tibshirani (1997) the risk of collision when using a cellphone was 4 time higher, RR = 4.3, 95%CI 3.0-6.5. Calls close to the time of collision has RR =4.8 for 5 minutes and RR = 5.9, $p<0.001$, for 15 minutes.

- Significant changes in local temperature, and in physiologic parameters of the CNS and cardiovascular system, Khdnisskii, Moshkarev and Fomenko (1999).

- Cardiac pacemaker interference: skipped three beats, Barbaro et al. (1996); showed interference, Hofgartner et al. (1996); significant interference, $p<0.05$ Chen et al. (1996); extremely highly significant interference, $p=0.0003$, Naegeli et al. (1996); $p<0.0001$, Altamura et al. (1997); reversible interference, Schlegal et al. (1998); significantly induced electronic noise, Occhetta et al. (1999); various disturbances observed and warnings recommended, Trigano et al. (1999)

- Increases blood pressure, Braune et al. (1998).

- Decreases in sperm counts and smaller tube development in rat testes, Dasdag et al. (1999).

- Increases embryonic mortality of chickens, Youbicier-Simo, Lebecq and Bastide (1998).

- Causes memory loss, concentration difficulties, fatigue, and headache, in a dose response manner, (Mild et al. (1998)). Headache, discomfort, nausea, Hocking (1998).

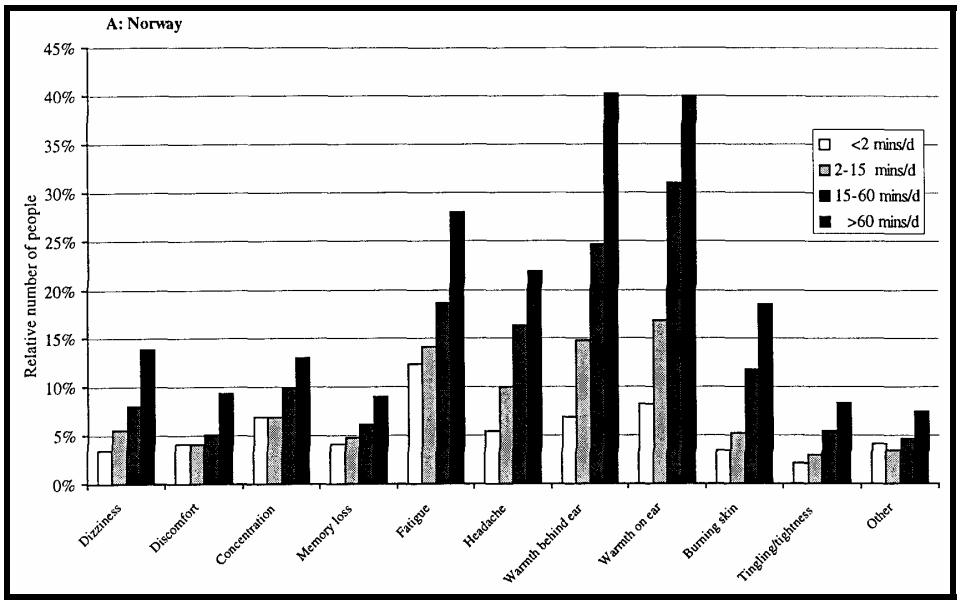

Figure 46: Prevalence of symptoms for Norwegian mobile phone users, mainly analogue, with various categories of length of calling time per day, Mild et al. (1998).

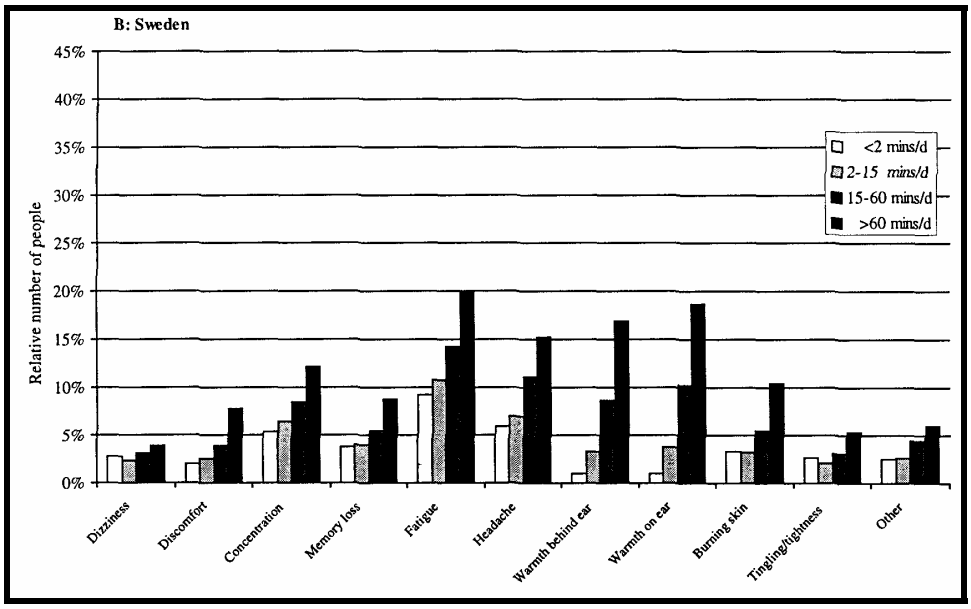

Figure 47: Prevalence of symptoms for Swedish mobile phone users, mainly digital, with various categories of length of calling time per day, Mild et al. (1998).

These are the same symptoms that have frequently been reported as "Microwave Sickness Syndrome" or "Radiofrequency Sickness Syndrome", Baranski and Czerski (1976) and Johnson-Liakouris (1998).

- Reduces the pituitary production of Thyrotropin (Thyroid Stimulating Hormone, TSH):

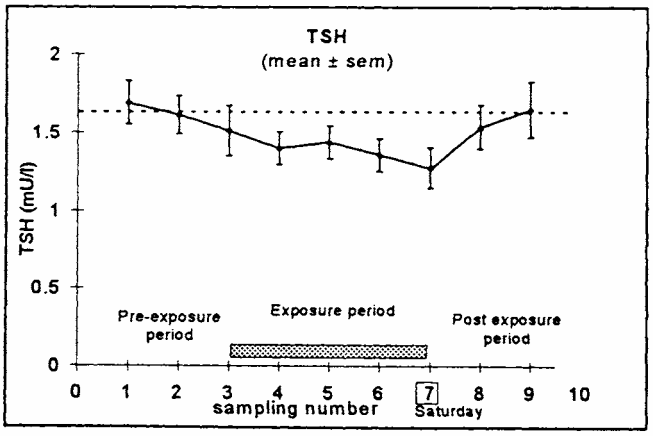

Figure 48: A significant reduction in Thyrotropin (Thyroid Stimulating Hormone) during cell phone use, de Seze et al. (1998).

- A reported but yet to be published Australian Study, EMRAA News, June 2000, used a Clot Retention Test on blood samples to detect hormonal changes. A group of 30 volunteers used a Nokia 6150 cellphone for 10 minutes on each of two consecutive days. The CRT test showed significant changes in the thyroid, pancreas, ovaries, testes and hormonal balance.

- Reduces melatonin, Burch et al. (1997, 1998).

- Breaks DNA strands (Verschaeve at al. (1994), Maes et al. (1997), which is still extremely significant $p<0.0001$, at 0.0024W/kg (1.2 $\mu W/cm^2$), Phillips et al. (1998)).

- Produces an up to three-fold increase in chromosome aberrations in a dose response manner from all cell phones tested, Tice, Hook and McRee, reported in Microwave News, April/May 1999.

- Doubles c-fos gene activity (a proto oncogene) for analogue phones and increases it by 41 % for digital phones, Goswami et al. (1999), altered c-jun gene, Ivaschuk et al. (1997), Increased hsp70 messenger RNA, Fritz et al. (1997).

- Increases Tumour Necrosis Factor (TNK), Fesenko et al. (1999).

- Increases ODC activity, Penafiel et al. (1997).

- DNA synthesis and cell proliferation increased after 4 days of 20 min for 3 times/day exposure. Calcium ions were significantly altered, French, Donnellan and McKenzie (1997). Decreased cell proliferation, Kwee and Raskmark (1997), Velizarov, Raskmark and Kwee (1999)

- Doubles the cancer in mice, Repacholi et al. (1997).

- Increases the mortality of users of older mobile phones that exposed the whole body, compared with users of more modern hand-held cellphone that primarily expose the head, RR = 1.38, 95%CI: 1.07-1.79, p=0.013, Rothman et al. (1996b).

- Increases human brain tumor rate by 2.5 times (Hardell et al. (1999a)).

- Associated with an angiosarcoma (case study), Hardell (1999b).

- Gandhi (1999) showed that cellphone radiation penetrated children's heads much more than adults.

- Muscat et al. (1999) found a significant increase of ganglioneuromas/ ganglioneuroblastomas in 14 out of 34 cellphone users.

- Hardell et al. (2000), for analogue phones OR = 2.62, 95%CI: 1.02-6.71, with higher tumour rates at points of highest exposure.

- Carlo and Jenrow (2000) report WTR research that shows micronuclei damage in human blood and an increase in brain tumor mortality among hand-held cellphone users compared with car phone users.

Conclusions:

To date 53 studies have shown adverse biological or human health effects specifically from cell phone radiation. These research results to date clearly show that cell phones and cell phone radiation are a strong risk factor for all of the adverse health effects identified for EMR because they share the same biological mechanisms. The greatest risk is to cell phone users because of the high exposure to their heads and the great sensitivity of brain tissue and brain processes. DNA damage accelerates cell death in the brain, advancing neurodegenerative diseases and brain cancer. Brain tumour is already an identified risk factor. Cell phones are carried on people's belts and in breast pockets. Hence liver cancer, breast cancer and testicular cancer became probable risk factors.

Altered attention and cognition, as well as the diversion of talking on a phone while driving is a significant risk factor for accidents and fatal accidents.

Some cardiac pacemakers are susceptible to active cell phone signals, recommending keeping cell phones away from hearts and pacemakers.

Because the biological mechanisms are shown and EMR has been observed to significantly increase the following effects, there is extremely strong evidence to conclude that cell phones are a risk factor for breast, liver, testicular and brain cancer. It is also probable that we will observe a very wide range of other effects including cardiac, neurological and reproductive illness and death. Since cell phone radiation cause many cell damages including interference with the brain's EEG and cardiac pace-makers, DNA and chromosome damage, all of these effects will also be caused by cell sites.

Recommendations:

Since the threshold level for no-effects is zero exposure, and since buildings can shield RF/Microwaves by at least a factor of 10, if a maximum exposure level at the boundary of a property is set at 0.1μW/cm^2, then the indoor exposure will be less than 0.01μW/cm^2, or 10 nW/cm^2. This reduces the health risk to less from outside than using a computer or a being in a kitchen with the microwave oven on. Hence I recommend an outdoor public exposure limit at the boundary of properties of 0.1μW/cm^2, the Salzburg cell site limit.

References:

Abelin, T., 1999: "Sleep disruption and melatonin reduction from exposure to a shortwave radio signal". Seminar at Canterbury Regional Council, New Zealand. August 1999.

Adey, W.R., 1980: “Frequency and Power windowing in tissue interactions with weak electromagnetic fields”. Proc. IEEE, 68:119-125.

Adey, W.R., 1981: "Tissue interactions with non-ionizing electromagnetic fields". Physiological Reviews 61: 435-514.

Adey, W.R., Byus, C.V., Cain, C.D., Higgins, R.J., Jones, R.A., Kean, C.J., Kuster, N, MacMurray, A., Stagg, R.B. and Zimmerman G., 2000: "Spontaneous and nitrosourea-induced primary tumors of the central nervous system in Fischer 344 rats exposed to frequency modulated microwaves". Cancer Research, 60(7): 1857-1863.

Alberts, B., Bray, D., Lewis, J., Raff, M., Roberts, K. and Watson, J.D., 1994: "Molecular Biology of the cell". 3rd edition, New York, Garland Publishing, 1994.

Ahissar, E., Haidarliu, S. and Zacksenhouse, M., 1997: "Decoding temporally encoded sensory input by cortical oscillations and thalamic phase comparators". Proc Nat Acad Sci USA 94:11633-11638.

Ahuja, Y.R., Bhargava, A., Sircar, S., Rizwani, W., Lima, S., Devadas, A.H. and Bhargava, S.C., 1997: "Comet assay to evaluate DNA damage caused by magnetic fields". Proceed. "International Conference on Electromagnetic Interference and compatability", Dec 3-5, 1997, Hyderbad, India.

Alberts, E.N., 1977: "Light and electron microscopic observations on the blood-brain barrier after microwave irradiation. In Symposium on Biological effects and measurement of Radio Frequency/Microwaves, HEW Publication (FDA) 77-8026, pp 294-309.

Alberts, E.N., 1978: "Reversibility of microwave induced blood-brain barrier permeability". Radio Science Supplement.

Altpeter, E.S., Krebs, Th., Pfluger, D.H., von Kanel, J., Blattmann, R., et al., 1995: "Study of health effects of Shortwave Transmitter Station of Schwarzenburg, Berne, Switzerland". University of Berne, Institute for Social and Preventative Medicine, August 1995.

Archimbaud, E., Charrin, C., Guyotat, D., and Viala, J-J, 1989: "Acute myelogenous leukaemia following exposure to microwaves". British Journal of Haematology, 73(2): 272-273.

Arnetz, B.B. and Berg, M., 1996: "Melatonin and Andrenocorticotropic Hormone levels in video display unit workers during work and leisure. J Occup Med 38(11): 1108-1110.

Altamura G, Toscano S, Gentilucci G, Ammirati F, Castro A, Pandozi C, Santini M, 1997: "Influence of digital and analogue cellular telephones on implanted pacemakers". Eur Heart J 18(10): 1632-4161.

Balcer-Kubiczek, E.K. and Harrison, G.H., 1991: "Neoplastic transformation of C3H/10T1/2 cells following exposure to 120Hz modulated 2.45 GHz microwaves and phorbol ester tumor promoter". Radiation Research, 125: 65-72.

Balode, Z., 1996: “Assessment of radio-frequency electromagnetic radiation by the micronucleus test in Bovine peripheral erythrocytes”. The Science of the Total Environment, 180: 81-86.

Balodis, V., Brumelis, G., Kalviskis, K., Nikodemus, O., Tjarve, D., and Znotina, V., 1996: ”Does the Skrunda Radio Location Station diminish the radial growth of pine trees?”. The Science of the Total Environment, Vol 180, pp 57-64.

Band, P.R., Le, N.D., Fang, R., Deschamps, R., Coldman, A., Gallagher, R.P. and Moody, J., 1996: "Cohort study of Air Canada pilots: mortality incidence and leukaemia risk. Am J Epidemiol 143(2):137-143.

Band, P.R., Spinelli, J.J., Ng, V.T., Moody, J. and Gallagher, R.P., 1990: "Mortality and cancer incidence in a cohort of commercial airline pilots". Aviat Space Environ Med 61(4): 299-302.

Barbaro V, Bartolini P, Donato A, Militello C, 1996: "Electromagnetic interference of analog cellular telephones with pacemakers". Pacing Clin Electrophysiol 19(10): 1410-1418.

Baranski, S. and Czerski, P., 1976: "Biological effects of microwaves". Publ. Dowden, Hutchison and Ross, Inc. Stroudsburg, Pennsylvania.

Baris, D. and Armstrong, B., 1990: "Suicide among electric utility workers in England and Wales". Br J Indust Med 47:788-789.

Barron, C.I. and Baraff, A.A, 1958: "Medical considerations of exposure to microwaves (Radar)". Journal American Medical Association, 168(9):1194-1199.

Bawin, S.M. and Adey, W.R., 1976: “Sensitivity of calcium binding in cerebral tissue to weak electric fields oscillating at low frequency”. Proc. Natl. Acad. Sci. USA, 73: 1999-2003.

Baris, D. and Armstrong, B., 1990: "Suicide among electric utility workers in England and Wales". Br J Indust Med 47:788-789.

Bawin, S.M. and Adey, W.R., 1976: “Sensitivity of calcium binding in cerebral tissue to weak electric fields oscillating at low frequency”. Proc. Natl. Acad. Sci. USA, 73: 1999-2003.

Beale, I.L., Pearce, N.E., Conroy, D.M., Henning, M.A., and Murrell, K., A., 1997: "Psychological effects of chronic exposure to 50 Hz magnetic fields in humans living near extra-high-voltage transmission lines". Bioelectromagnetics, 18(8): 584-94.

Beall, C., Delzell, E., Cole, P., and Brill, I., 1996: "Brain tumors among electronics industry workers". Epidemiology, 7(2): 125-130.

Belanger, K., Leaderer, B., Hellenbrand, K., Holford, T.R., McSharry, J-E., Power, M-E, and Bracken, M.B., 1998: "Spontaneous abortion and exposure to electric blankets and heated water beds". Epidemiology, 9: 36-42.

Berman, E., Carter, H.B., and House D., 1982: “Reduce weight in mice offspring after in utero exposure to 2450 MHz (CW) microwaves”. Bioelectromagnetics, 3(2): 285-291.

Blackman, C.F., Benane, S.G., Elliott, D.J., and Pollock, M.M., 1988: “Influence of Electromagnetic Fields on the Efflux of Calcium Ions from Brain Tissue in Vitro: A Three-Model Analysis Consistent with the Frequency Response up to 510 Hz”. Bioelectromagnetics, 9:215-227.

Blackman, C.F., Kinney, L.S., House, D.E., and Joines, W.T., 1989: “Multiple power-density windows and their possible origin”. Bioelectromagnetics, 10: 115-128.

Blackman, C.F., 1990: "ELF effects on calcium homeostasis". In "Extremely low frequency electromagnetic fields: The question of cancer", BW Wilson, RG Stevens, LE Anderson Eds, Publ. Battelle Press Columbus: 1990; 187-208.

Blackman, C.F., Benane, S.G., and House, D.E., 1991: “The influence of temperature during electric- and magnetic-field induced alteration of calcium-ion release from in vitro brain tissue”. Bioelectromagnetics, 12: 173-182.

Borbely, AA, Huber, R, Graf, T, Fuchs, B, Gallmann, E, Achermann, P, 1999: Pulsed high-frequency electromagnetic field affects human sleep and sleep electroencephalogram. Neurosci Lett 275(3):207-210.

Bortkiewicz, A., Zmyslony, M., Palczynski, C., Gadzicka, E. and Szmigielski, S., 1995: "Dysregulation of autonomic control of cardiac function in workers at AM broadcasting stations (0.738-1.503 MHz)". Electro- and Magnetobiology 14(3): 177-191.

Bortkiewicz, A., Gadzicka, E. and Zmyslony, M., 1996: "Heart rate in workers exposed to medium-frequency electromagnetic fields". J Auto Nerv Sys 59: 91-97.

Bortkiewicz, A., Zmyslony, M., Gadzicka, E., Palczynski, C. and Szmigielski, S., 1997: "Ambulatory ECG monitoring in workers exposed to electromagnetic fields". J Med Eng and Tech 21(2):41-46.

Braune, S, Wrocklage, C, Raczek, J, Gailus, T, Lucking, CH, 1998: Resting blood pressure increase during exposure to a radio-frequency electromagnetic field. Lancet 351(9119):1857-1858.

Brown-Woodman, P.D., Hadley, J.A., Richardson, L., Bright, D. and Porter, D., 1989: "Evaluation of reproductive function of female rats exposed to radiofrequency fields (27.12 MHz) near a shortwave diathermy device". Health Physics 56(4): 521-525.

Brueve, R., Feldmane, G., Heisele, O., Volrate, A. and Balodis, V., 1998: "Several immune system functions of the residents from territories exposed to pulse radio-frequency radiation". Presented to the Annual Conference of the ISEE and ISEA, Boston Massachusetts July 1998.

Burch, J.B., Reif, J.S., Pittrat, C.A., Keefe, T.J. and Yost, M.G., 1997: "Cellular telephone use and excretion of a urinary melatonin metabolite". In: Annual review of Research in Biological Effects of electric and magnetic fields from the generation, delivery and use of electricity, San Diego, CA, Nov. 9-13, P-52.

Burch, J.B., Reif, J.S., Yost, M.G., Keefe, T.J. and Pittrat, C.A., 1998: "Nocturnal excretion of urinary melatonin metabolite among utility workers". Scand J Work Environ Health 24(3): 183-189.

Burch, J.B., Reif, J.S., Yost, M.G., Keefe, T.J. and Pittrat, C.A., 1999a: "Reduced excretion of a melatonin metabolite among workers exposed to 60 Hz magnetic fields" Am J Epidemiology 150(1): 27-36.

Burch, J.B., Reif, J.S. and Yost, M.G., 1999b: "Geomagnetic disturbances are associated with reduced nocturnal excretion of melatonin metabolite in humans". Neurosci Lett 266(3):209-212.

Burch, J.B., Reif, J.S., Noonan, C.W. and Yost, M.G., 2000: "Melatonin metabolite levels in workers exposed to 60-Hz magnetic fields: work in substations and with 3-phase conductors". J of Occupational and Environmental Medicine, 42(2): 136-142.

Byus, C.V., Kartun, K., Pieper, S. and Adey, W.R., 1988: "Increased ornithine decarboxylase activity in cultured cells exposed to low energy modulated microwave fields and phorbol ester tumor promoters". Cancer research, 48(15): 4222-4226.

Burke, J., 1985: "The Day the Universe Changed". Publ. Little and Co., Boston.

Campbell-Beachler, M., Ishida-Jones, T., Haggren, W. and Phillips, J.L., 1998: "Effect of 60 Hz magnetic field exposure on c-fos expression in stimulated PC12 cells". Mol Cell Biochem 189(1-2): 107-111.

Cantor, K.P., Stewart, P.A., Brinton, L.A., and Dosemeci, M., 1995: "Occupational exposures and female breast cancer mortality in the United States". Journal of Occupational Medicine, 37(3): 336-348.

Carlo, L.G. and Jenrow, R.S., 2000: "Scientific Progress- Wireless Phones and Brain Cancer: Current state of the science". MedGenMed, July 31, 2000. www.medscape.com/Medscape/GeneralMedicine/journal/2000/v02.n04/mgm0731.carl/mgm0731.html

Capone, G., Choi, C. and Vertifuille, J., 1998: "Regulation of the prepromsomatostatin gene by cyclic-AMP in cerebrocortical neurons". Bran Res Mol Brain Res 60(2): 247-258.

Catterall, W.A., 1992: "Cellular and molecular biology of voltage-gated sodium channels". Physiological Reviews 72(4): S15-S48.

Chazan, B., Janiak, M., Szmigielski, S., and Troszynski, M., 1983: "Development of murine embryos and fetuses after irradiation with 2450 MHz microwaves". Problemy Medycyny Wieku Rozwojowego,12:164-173.

Chen WH, Lau CP, Leung SK, Ho DS, Lee IS, 1996: "Interference of cellular phones with implanted permanent pacemakers". Clin Cardiol 19(11): 881-886.

Cherry, N.J., 2000: "Evidence that Electromagnetic Radiation is Genotoxic - The implications for the epidemiology of cancer and cardiac, neurological and reproductive effects". Proceedings: Health Effects of Electromagnetic Radiation Conference. European Parliament, June 28, 2000.

Cherry, N.J., 2000a: Re: Cancer incidence near Radio and Television Transmitters in Great Britain, Dolk et al. (1997a,b)": Letter: American Journal of Epidemiology, (In Press) 2000.

Chou, C-K., Guy, A.W., Kunz, L.L., Johnson, R.B., Crowley, J.J. and Krupp, JH., 1992: "Long-term, low-level microwave irradiation of rats". Bioelectromagnetics 13: 469-496.

Cossarizza, A., Angioni, S., Petraglia, F., Genazzani, A.R., Monti, D., Capri, M., Bersani, F., Cadossi, R. and Franceschi, C., 1993: "Exposure to low frequency pulsed electromagnetic fields increases interleukin-1 and interleukin-6 production by human peripheral blood mononuclear cells". Exp Cell Res 204(2):385-387.

Cox, B., 1995: "Projections of the cancer burden in New Zealand". Public Health Commission, Wellington New Zealand.

Daniells, C, Duce, I, Thomas, D, Sewell, P, Tattersall, J, de Pomerai, D, 1998: "Transgenic nematodes as biomonitors of microwave-induced stress". Mutat Res 399: 55-64.

Dasdag, S, Ketani, MA, Akdag, Z, Ersay, AR, Sar,i I, Demirtas ,OC, Celik, MS, 1999: Whole-body microwave exposure emitted by cellular phones and testicular function of rats. Urol Res 27(3):219-223.

Davidson, J.A., 1998: "Brain tumours and mobile phones?". Medical Journal of Australia 168:48

Davis, S., 1997: "Weak residential Magnetic Fields affect Melatonin in Humans", Microwave News, Nov/Dec 1997.

Davis, R.L. and Mostofl, 1993: "Cluster of testicular cancer in police officers exposed to hand-held radar". Am. J. Indust. Med. 24: 231-233.

De Guire, L., Theriault, G., Iturra, H., Provencher, S., Cyr, D., and Case, B.W., 1988: "Increased incidence of malignant melanoma of the skin in workers in a telecommunications industry". British Journal of Industrial Medicine, Vol 45, pp 824-828.

De Mattei, M., Caruso, A., Traina, G.C., Pezzetti, F., Baroni, T., and Sollazzo, V., 1999: "Correlation between pulsed electromagnetic fields exposure time and cell proliferation increase in human osteosarcoma cell lines and human normal osteoblast cells in vitro". Bioelectromagnetics 20: 177-182.

De Pomerai, D., Daniells, C., David, H., Duce, I., Mutwakil, M., Thomas, D., Sewell, P., Tattersall, J., Jones, D., and candido, P., 2000: "Non-thermal heat-shock response to microwaves". Nature May 25,

Deroche, M., 1971: " Etude des perturbations biologiques chez les techniciens O.R.T.F. dans certains champs electromagnetiques de haute frequence". Arch Mal. Prof, 32: 679-683.

de Seze R, Fabbro-Peray P, Miro L, 1998: GSM radiocellular telephones do not disturb the secretion of antepituitary hormones in humans. Bioelectromagnetics 19(5):271-8.

Dmoch, A. and Moszczynski, P., 1998: "Levels of immunoglobulin and subpopulations of T lymphocytes and NK cells in men occupationally exposed to microwave radiation in frequencies of 6-12GHz". Med Pr 49(1):45-49.

Dolk, H., Shaddick, G., Walls, P., Grundy, C., Thakrar, B., Kleinschmidt, I. and Elliott, P., 1997a: “Cancer incidence near radio and television transmitters in Great Britain, I - Sutton-Colfield transmitter”. American J. of Epidemiology, 145(1):1-9.

Dolk, H., Elliott, P., Shaddick, G., Walls, P., Grundy, C., and Thakrar, B.,1997b: “Cancer incidence near radio and television transmitters in Great Britain, II All high power transmitters”. American J. of Epidemiology, 145(1):10-17.

Doll, R. and Hill A.B., 1964: British Medical Journal, i, 1399-1460.

Donnellan M, McKenzie DR, French PW, 1997: Effects of exposure to electromagnetic radiation at 835 MHz on growth, morphology and secretory characteristics of a mast cell analogue, RBL-2H3. Cell Biol Int 21:427-439.

El Nahas, S.M. and Oraby, H.A., 1989: "Micronuclei formation in somatic cells of mice exposed to 50 Hz electric fields. Environ Mol Mutagen 13(2):107-111.

Elwood, J.M., 1999: "A critical review of epidemiologic studies of Radiofrequency exposure and Human cancers". Env. Health. Persp., 107(Supplement 1) February 1999.

Eulitz, C, Ullsperger, P, Freude, G, Elbert ,T, 1998: Mobile phones modulate response patterns of human brain activity. Neuroreport 9(14):3229-3232.

Evans, J.A., Savitz, D.A., Kanal, E. and Gillen, J., 1993: "Infertility and pregnancy outcome among magnetic resonance imaging workers". J Occup Med 35(12): 1191-1195.

Fesenko, EE, Makar, VR, Novoselova, EG, Sadovnikov, VB, 1999: Microwaves and cellular immunity. I. Effect of whole body microwave irradiation on tumor necrosis factor production in mouse cells. Bioelectrochem Bioenerg 49(1):29-35.

Forman, S.A., Holmes, C.K., McManamon, T.V., and Wedding, W.R., 1982: "Physiological Symptoms and Intermittent Hypertension following acute microwave exposure". J. of Occup. Med. 24(11): 932-934.

Frei, M.R., Jauchem, J.R., Dusch, S.J., Mettitt, J.H., Berger, R.E. and Stedham, M.A., 1998: "chronic, low-level (1.0W/kg) exposure of mice prone to mammary cancer to 2450 MHz microwaves". Radiation Research 150(5): 568-576.

Freude, G, Ullsperger, P, Eggert ,S, Ruppe, I, 1998: Effects of microwaves emitted by cellular phones on human slow brain potentials. Bioelectromagnetics 19(6):384-387.

French PW, Donnellan M, McKenzie DR, 1997: Electromagnetic radiation at 835 MHz changes the morphology and inhibits proliferation of a human astrocytoma cell line. Bioelectrochem Bioenerg 43:13-18.

Freude, G, Ullsperger, P, Eggert, S, Ruppe, I, 2000: Microwaves emitted by cellular telephones affect human slow brain potentials. Eur J Appl Physiol 81(1-2):18-27.

Frey, A.H., Feld, S.R. and Frey. B., 1975: "Neural function and behavior: defining the relationship in biological effects of nonionizing radiation". Ann. N.Y. Acad. Sci. 247: 433-438.

Feychting, M., Schulgen, G., Olsen, J.H., and Ahlbom, A., 1995: "Magnetic fields and childhood cancer-pooled analysis of two Scandinavian studies". European J. of Cancer, 31A (12): 2035-2039.

Feychting, M, Schulgen, G., Olsen ,J.H. and Ahlbom, A., 1995: "Magnetic fields and childhood cancer - a pooled analysis of two Scandinavian studies". Eur. J. Cancer 31A(12): 2035-2039.

Forman, S.A., Holmes, C.K., McManamon, T.V., and Wedding, W.R., 1982: "Physiological Symptoms and Intermittent Hypertension following acute microwave exposure". J. of Occup. Med. 24(11): 932-934.

Frey, A.H., 1993: "Electromagnetic field interactions with biological systems". FASEB J., 7: 272-281.

Frey, A.H., 1995: "An integration of the data on mechanisms with particular reference to cancer", Chapter 2 in "On the Nature of electromagnetic Field Interactions with Biological Systems", Ed A.H. Frey, Publ. R.G. Landes Co. Medical Intelligence Unit, Austin, Texas.

Frey, A.H., 1998: "Headaches from cellular telephones: are they real and what are the impacts". Environ Health Perspect 106(3):101-103.

Fritze K, Wiessner C, Kuster N, Sommer C, Gass P, Hermann DM, Kiessling M, Hossmann KA, 1997: Effect of global system for mobile communication microwave exposure on the genomic response of the rat brain. Neuroscience 81(3):627-639.

Gadzicka, E., Bortkiewicz, A., Zmyslony, M. and Palczynski, C., 1997: Evaluation of selected functional circulation parameters of workers from occupational groups exposed to electromagnetic fields of high frequency. III 24-h monitoring of arterial blood pressure". Med Pr, 48(1): 15-24.

Garcia-Sagredo, J.M. and Monteagudo, J.L., 1991: "Effect of low-level pulsed electromagnetic fields on human chromosomes in vitro: analysis of chromosome aberrations". Hereditas 115(1): 9-11.

Garaj-Vrhovac, V., Fucic, A, and Horvat, D., 1990: "Comparison of chromosome aberration and micronucleus induction in human lymphocytes after occupational exposure to vinyl chloride monomer and microwave radiation"., Periodicum Biologorum, Vol 92, No.4, pp 411-416.

Garaj-Vrhovac, V., Horvat, D. and Koren, Z., 1991: "The relationship between colony-forming ability, chromosome aberrations and incidence of micronuclei in V79 Chinese Hamster cells exposed to microwave radiation". Mutat Res 263: 143-149.

Garaj-Vrhovac, V., Fucic, A, and Horvat, D., 1992: The correlation between the frequency of micronuclei and specific aberrations in human lymphocytes exposed to microwave radiation in vitro". Mutation Research, 281: 181-186.

Garaj-Vrhovac, V., and Fucic, A., 1993: "The rate of elimination of chromosomal aberrations after accidental exposure to microwave radiation". Bioelectrochemistry and Bioenergetics, 30:319-325.

Garaj-Vrhovac, V., 1999: "Micronucleus assay and lymphocyte mitotic activity in risk assessment of occupational exposure to microwave radiation. Chemosphere 39(13): 2301-2312.

Garaj-Vrhovac, V., Fucic, A, and Horvat, D., 1990: "Comparison of chromosome aberration and micronucleus induction in human lymphocytes after occupational exposure to vinyl chloride monomer and microwave radiation"., Periodicum Biologorum, Vol 92, No.4, pp 411-416.

Gey, K.F., 1993: "Prospects for the prevention of free radical disease, regarding cancer and cardiovascular disease". British Medical Bulletin, 49(3): 679-699.

Goldsmith, J.R., 1995: "Epidemiological Evidence of Radiofrequency Radiation (Microwave) Effects on Health in Military, Broadcasting, and Occupational Studies". International Journal of Occupational and Environmental Health, 1, pp 47-57, 1995.

Goldsmith, J.R., 1996: "Epidemiological studies of radio-frequency radiation: current status and areas of concern". The Science of the Total Environment, 180: 3-8.

Goldsmith, J.R., 1997: "TV Broadcast Towers and Cancer: The end of innocence for Radiofrequency exposures". Am. J. Industrial Medicine 32 : 689-692.

Goldsmith, J.R., 1997a: "Epidemiologic evidence relevant to radar (microwave) effects". Environmental Health Perspectives, 105 (Suppl 6): 1579-1587.

Gordon, Z.V., 1966: "Problems of industrial hygiene and the biological effects of electromagnetic superhigh frequency fields". Moscow Medicina [In Russian] English translation in NASA Rept TT-F-633, 1976.

Goodman, R., Wei., L.X., Xu, J.C. and Henderson, A., 1989: "Exposure of human cells to low-frequency electromagnetic fields results in quantitative changes in transcripts". Biochim Biophys Acta 1009(3): 216-220.

Goodman, R., Weisbrot, D., Uluc, A. and Henderson A., 1992: "Transcription in Drosophila melanogaster salivary cells is altered following exposure to low-frequency electromagnetic fields: analysis of chromosome 3R". Bioelectromagnetics 13(2): 111-118.

Goswami, P.C., Albee, L.D., Parsian, A.J., Baty, J.D., Moros, E.G., Pickard, W.F., Roti Roti, J.L. and Hunt, C.R., 1999: "Proto-oncogene mRNA levels and activities of multiple transcription factors in C3H 10T 1/2 murine embryonic fibroblasts exposed to 835.62 and 847.74 MHz cellular telephone communication frequency radiation". Radiat Res 151(3): 300-309.

Graham, C., Cook, M.R., Cohen, H.D. and Gerkovich, M.M., 1994: "A dose response study of human exposure to 60Hz electric and magnetic fields". Bioelectromagnetics 15: 447-463.

Graham, C., Cook, M.R., Sastre, A., Riffle, D.W. and Gerkovich, M.M., 2000: "Multi-night exposure to 60 Hz magnetic fields: effects on melatonin and its enzymatic metabolite". J Pineal Res 28(1): 1-8.

Grandolfo, M., Michaelson, S.M. and Rindi, A. (Eds), 1985: "Biological effects and dosimetry of static and ELF electromagnetic fields". New York, Plenum Press.

Grayson, J.K., 1996: “Radiation Exposure, Socioeconomic Status, and Brain Tumour Risk in the US Air Force: A nested Case-Control Study”. American J. of Epidemiology, 143 (5), 480-486.

Haider, T., Knasmueller, S., Kundi, M, and Haider, M., 1994: “Clastogenic effects of radiofrequency radiation on chromosomes of Tradescantia”. Mutation Research, 324:65-68.

Hamburger, S., Logue, J.N., and Sternthal, P.M., 1983: "Occupational exposure to non-ionizing radiation and an association with heart disease: an exploratory study". J Chronic Diseases, Vol 36, pp 791-802.

Hammett and Edison Inc., 1997: "Engineering analysis of radio frequency exposure conditions with addition of digital TV channels". Prepared for Sutra Tower Inc., San Francisco, California, January 3, 1997.

Hansson Mild, K, Oftedal, G, Sandstrom, M, Wilen, J, Tynes, T, Haugsdal, B, Hauger E, 1998: Comparison of symptoms experienced by users of analogue and digital mobile phones: a Swedish-Norwegian epidemiological study. Arbetslivsrapport 23.

Hardell, L., Holmberg, B., Malker, H., and Paulsson, L.E., 1995: "Exposure to extremely low frequency electromagnetic fields and the risk of malignant diseases--an evaluation of epidemiological and experimental findings". Eur. J. Cancer Prevention, 1995 Sep;4 Suppl 1:3-107

Hardell, L, Reizenstein, J, Johansson, B, Gertzen, H, Mild, KH, 1999: Angiosarcoma of the scalp and use of a cordless (portable) telephone. Epidemiology 10(6):785-786.

Hardell, L, Nasman, A, Pahlson, A, Hallquist, A, Hansson Mild, K, 1999: Use of cellular telephones and the risk for brain tumours: A case-control study. Int J Oncol 15(1):113-116.

Hardell, L, Nasman, A, Hallquist, A, 2000: "Case-control study of radiology work, medical X-ray investigations and use of cellular telephones as risk factors". J of General Medicine. <www.medscape.com/Medscape/GeneralMedicine/journal/2000/v02.n03/>

Hayes, R.B., Morris Brown, L., Pottern, L.M., Gomez, M., Kardaun, J.W.P.F., Hoover, R.N., O'Connell, K.J., Sutsman, R.E. and Nasser, J., 1990: Occupational and Risk for Testicular Cancer: A Case Control Study. International Journal of Epidemiology, 19, No.4, pp 825-831.

Heller, J.H., and Teixeira-Pinto, A.A., 1959: “A new physical method of creating chromosome aberrations”. Nature, Vol 183, No. 4665, March 28, 1959, pp 905-906.

Hietanen, M., Kovala, T. and Hamalainem, A.M., 2000: "Human brain activity during exposure to radio frequency fields emitted by cellular phones". Scand J Work Environ Health 26(2): 87-92.

Hill, A. B., 1965: “The Environment and Disease: Association or Causation?" Proc. Royal Society of Medicine (U.K.). 295-300.

Hladky, A, Musil, J, Roth, Z, Urban, P, Blazkova, V, 1999: Acute effects of using a mobile phone on CNS functions. Cent Eur J Public Health 7(4):165-167.

Hocking, B. and Joyner, K., 1995: “Re: Miscarriages among Female Physical Therapists who report using radio- and microwave- frequency electromagnetic radiation.” - A letter to the Editor, American J. of Epidemiology, 141 (3): 273-274.

Hocking, B., Gordon, I.R., Grain, H.L., and Hatfield, G.E., 1996: “Cancer incidence and mortality and proximity to TV towers”. Medical Journal of Australia, 165: 601-605.

Hocking, B., 1998: Comment on "Brain tumours and mobile phones?". Medical Journal of Australia 168:48

Hocking, B, 1998: Preliminary report: symptoms associated with mobile phone use. Occup Med (Lond);48(6):357-360.

Hocking, B., Gordon, I.R. and Hatfield, G.E., 1999: Childhood leukaemia and TV Towers revisited". Australian and New Zealand J. Public Health, 23(1): 104-105.

Hocking, B. and Gordon, I.R., 2000: "Decreased survival for childhood leukaemia in proximity to TV towers". Poster presented at the Annual Scientific meeting of the Royal Australasian College of Physicians in Adelaide SA, 2-5 May 2000.

Hofgartner F, Muller T, Sigel H, 1996: "Could C- and D-network mobile phones endanger patients with pacemakers?". Dtsch Med Wochenschr 121(20): 646-652,. [Article in German]

ICNIRP (1998): "Guidelines for limiting exposure to time-varying electric, and electromagnetic fields (up to 300 GHz) - ICNIRP Guidelines". International Commission on Non-Ionizing Radiation Protection Health Physics, 74(4):494-522.

IRPA, 1988: "Guidelines on limits of exposure to radiofrequency electromagnetic fields in the frequency range from 100 kHz to 300 GHz". Health Physics, 54(1): 115-123.

IRPA/INIRC 1990: "Interim guidelines on limits of exposure to 50/60 Hz electric and magnetic fields". Health Physics 58(1): 113-122.

Ivaschuk, O.I., Jones, R.A., Ishida-Jones, T., Haggren, Q., Adey, W.R. and Phillips, J.L., 1997: "Exposure of nerve growth factor-treated PC12 rat pheochromscytoma cells to a modulated radiofrequency field at 836.55 MHz: effects on c-jun and c-fos expression". Bioelectromagnetics 18(3): 223-229.

Jacobson, C.B., 1969: Progress report on SCC 31732, (Cytogenic analysis of blood from the staff at the U.S. Embassy in Moscow), George Washington University, Reproductive Genetics Unit, Dept. of Obstertics and Genocolgy, February 4, 1969.

Johanson C, Kock-Henriksen N, Rasmussen S, Olsen JH. 1999: "Multiple Sclerosis among utility workers". Neurology 52: 1279-1282.

Johnson-Liakouris, A.J.. 1998: "Radiofrequency (RF) Sickness in the Lilienfeld Study: an effect of modulated microwaves". Arch Environ Heath 53(3):236-238.

Jones, L.F., 1933: "A study of the propagation of wavelengths between three and eight meters. Proc. of the Institute of Radio Engineers 21(3): 349-386.

Jordan, E.C., (Ed), 1985: "Reference data for engineeres: Radio, Electronics, Computer and Communications, 7th Edition". Publ. Howard W. Sams & CO., Indianapolis.

Juutilainen, J., Matilainen, P., Saarikoski, S., Laara, E. and Suonio, S., 1993: "Early pregnancy loss and exposure to 50 Hz magnetic fields". Bioelectromagnetics, 14(3): 229-236.

Juutilainen, J., Stevens, R.G., Anderson, L.E., Hansen, N.H., Kilpelainen, M., Laitinen, J.T., Sobel, E. and Wilson, B.W., 2000: "Nocturnal 6-hydroxymelatonin sulphate excretion in female workers exposed to magnetic fields". J Pineal Res 28(2): 97-104.

Kallen, B., Malmquist, G., and Moritz, U., 1982: “Delivery Outcome among Physiotherapists in Sweden: is Non-ionizing Radiation a Fetal Hazard? Archives of Environmental Health, 37(2): 81-84.

Kalnins, T., Krizbergs, R., and Romancuks, A.,1996: “Measurement of the intensity of electromagnetic radiation from the Skrunda radio location station, Latvia”. The Science of the Total Environment, Vol 180, pp 51-56.

Kaplan, S., Etlin, S., Novkov, I, and Modan, B., 1997: "Occupational risks for the development of brain tumors. Am. J. Ind. Med. 31: 15-20.

Karasek, M., Woldanska-Okonska, M., Czernicki, J., Zylinska, K. and Swietoslawski, J., 1998: "Chronic exposure to 2.9 mT, 40 Hz magnetic field reduces melatonin concentrations in humans". J Pineal Research 25(4): 240-244.

Kellenyi, L, Thuroczy, G, Faludy, B, Lenard, L, 1999: Effects of mobile GSM radiotelephone exposure on the auditory brainstem response (ABR). Neurobiology 7:79-81.

Khaili, A.M. and Qassem, W., 1991: "Cytogenetic effects of pulsing electromagnetic field on human lymphocytes in vitro: chromosome aberrations, sister-chromatid exchanges and cell kinetics". Mutat Res 247: 141-146.

Khudnitskii, SS, Moshkarev, EA, Fomenko, TV, 1999: [On the evaluation of the influence of cellular phones on their users]. [Article in Russian] Med Tr Prom Ekol (9):20-24.

Kolomytkin, O., Kuznetsov, V., Yurinska, M, Zharikova, A., and Zharikov, S., 1994: “Response of brain receptor systems to microwave energy exposure”. pp 195-206 in “On the nature of electromagnetic field interactions with biological systems”, Ed Frey, A.H., Publ. R.G. Landes Co.

Koivisto, M, Revonsuo, A, Krause, C, Haarala, C, Sillanmaki, L, Laine, M, Hamalainen, H, 2000: Effects of 902 MHz electromagnetic field emitted by cellular telephones on response times in humans. Neuroreport 11(2):413-415.

Kolodynski, A.A. and Kolodynska, V.V., 1996: “Motor and psychological functions of school children living in the area of the Skrunda Radio Location Station in Latvia”. The Science of the Total Environment, Vol 180, pp 87-93.

Kolomytkin, O., Kuznetsov, V., Yurinska, M, Zharikova, A., and Zharikov, S., 1995: “Response of brain receptor systems to microwave energy exposure”. pp 195-206 in “On the nature of electromagnetic field interactions with biological systems”, Ed Frey, A.H., Publ. R.G. Landes Co.

König HL. 1974, Behavioural changes in human subjects associated with ELF electric fields. In Persinger MA, editor. ELF and VLF electromagnetic field effects. New York, Plenum Press.

Krause, C.M., Sillanmaki, L., Koivisto, M., Haggqvist, A., Saarela, C., Revonsuo, A., Laine, M. and Hamalainen H., 2000: "Effects of electromagnetic field emitted by cellular phones on the EEG during a memory task". Neuroreport 11(4): 761-764.

Kromhout, H., Loomis, D.P., Mihlan, G.J., Peipins, L.A., Kleckner, R.C., iriye, R. and Savitz, D.A., 1995: Assessment and grouping of occupational magnetic field exposure in five electric utility companies". Scand J Work Environ Health 21(1): 43-50.

Kwee, S, Raskmark, P, 1997: Radiofrequency electromagnetic fields and cell proliferation. Presented at the Second World Congress for Electricity and Magnetism in Biology and Medicine, Bologna, Italy, June.

Lagroye, I, and Poncy J.L., 1998: "Influences of 50 Hz magnetic fields and ionizing radiation on c-jun and c-fos oncoproteins". Bioelectromagnetics 19(2): 112-116.

Lai, H. and Singh, N.P., 1995: “Acute low-intensity microwave exposure increases DNA single-strand breaks in rat brain cells”. Bioelectromagnetics 16: 207-210.

Lai, H. and Singh, N.P., 1996: “Single- and double-strand DNA breaks in rat brain cells after acute exposure to radiofrequency electromagnetic radiation”. Int. J. Radiation Biology, 69 (4): 513-521.

Lai, H. and Singh, N.P., 1996a: "Reply to "Comment on 'Acute low-intensity microwave exposure increases DNA single-strand breaks in rat brain cells' ". Bioelectromagnetics 17: 166.

Lai, H., and Singh, N.P., 1997a: “Melatonin and N-tert-butyl-a-phenylnitrone Block 60 Hz magnetic field-induced DNA single- and double-strands Breaks in Rat Brain Cells.” Journal of Pineal Research 22:152-162.

Lai, H., and Singh, N.P., 1997b: “Melatonin and Spin-Trap compound Block Radiofrequency Electromagnetic Radiation-induced DNA Strands Breaks in Rat Brain Cells.” Bioelectromagnetics 18:446-454.

Lamble D, Kauranen T, Laakso M, Summala H, 1999: "Cognitive load and detection thresholds in car following situations: safety implications for using mobile (cellular) telephones while driving". Accid Anal Pre ;31(6):617-623.

Lancranjan, I.,Maicanescu, M., Rafaila, E., Klespsch, I. and Popescu, H.I., 1975: "Gonadic function in workmen with long-term exposure to microwaves". Health Physics, 29(3): 381-383.

Larsen, A.I., 1991: "Congenital malformations and exposure to high-frequency electromagnetic radiation among Danish physiotherapists" Sacand J work Environ Health 17(5): 318-323.

Larsen, A.I., Olsen, J., and Svane, O., 1991: "Gender specific reproductive outcome and exposure to high frequency electromagnetic radiation among physiotherapists". Scand. J. Work Environ. Health, Vol.17, pp 324-329.

Lester, J.R., and Moore, D.F., 1982a: “Cancer incidence and electromagnetic radiation”. Journal of Bioelectricity, 1(1):59-76.

Lester, J.R., and Moore, D.F., 1982b: “Cancer mortality and air force bases”. Journal of Bioelectricity, 1(1):77-82.

Lester, J.R., 1985: “Reply to: Cancer mortality and air force bases, a reevaluation”. Journal of Bioelectricity, 4(1):129-131.

Li, C.M., Chiang, H., Fu, Y.D., Shao, B.J., Shi, J.R. and Yao, G.D., 1999: "Effects of 50Hz magnetic fields on gap junction intercellular communication". Bioelectromagnetics 20(5):290-294.

Libersat, F., Levy, A. and Camhi, J.M., 1989: "Multiple feedback loops in the flying cockroach: excitation of the dorsal and inhibition of the ventral giant interneurons". J. Comp. Physiolo. 165(5): 651-668.

Liburdy, R.P., Callahan, D.E., Harland, J., Dunham, E., Sloma, T.R. and Yaswen, P., 1993: "Experimental evidence for 60 Hz magnetic fields operating through the signal transduction cascade - effects on calcium influx and c-MYC mRNA induction". FEBS Lett 334(3): 301-308.

Lilienfeld, A.M., Tonascia, J., and Tonascia S., Libauer, C.A., and Cauthen, G.M., 1978: “Foreign Service health status study - evaluation of health status of foreign service and other employees from selected eastern European posts”. Final Report (Contract number 6025-619073) to the U.S. Dept of State, July 31, 1978.

Lilienfeld, A.M., 1983: "Practical limitations of epidemiologic method". Environmental Health Perspectives, 52:3-8.

Lin, H., Goodman, R. and Shirley-Henderson, A., 1994: "Specific region of the c-myc promoter is responsible for electric and magnetic fields". J Cell Biochem 54 30: 281-288.

Lindbohm, M-L,, Hietanen, M., Kyyronen, P., Sallmen, M., von Nandelstadh, P., Taskinen, H., Pekkarinen, M., Ylikoski, M. and Hemminki, K., 1992: "Magnetic fields of video display terminals and spontaneous abortion". Am J Epidemiol 136:1041-1051.

Litovitz, T.A., Montrose, C.J., Goodman, R. and Elson, E.C., 1990: "Amplitude windows and transiently augmented transcription from exposure to electromagnetic fields". Bioelectromagnetics 11(4): 297-312.

Litovitz, T.A., Krause, D., Penafiel, M., Elson, E.C. and Mullins, J.M., 1993: "The role of coherence time in the effect of microwaves on ornithine decarboxylase activity". Bioelectromagnetics 14(5): 395-403.

London, S.J., Thomas, D.C., Bowman, J.D., Sobel, E., Chen, T.S. and Peters J.M., 1991: "Exposure to residential electric and magnetic fields and risk of childhood leukemia". Am. J. Epidemiology 134 (9): 923-937.

Maskarinec, G., and Cooper, J., 1993: "Investigation of a childhood leukemia cluster near low-frequency radio towers in Hawaii". SER Meeting, Keystone, Colorado, June 16-18, 1993. Am. J. Epidemiology, 138:666, 1993.

Maes, A., Verschaeve, L., Arroyo, A., De Wagter, C. and Vercruyssen, L., 1993: "In vitro effects of 2454 MHz waves on human peripheral blood lymphocytes". Bioelectromagnetics 14: 495-501.

Maes, A., Collier, M., Slaets, D., and Verschaeve, L., 1996: "954 MHz Microwaves enhance the mutagenic properties of Mitomycin C". Environmental and Molecular Mutagenesis, 28: 26-30.

Maes A, Collier M, Van Gorp U, Vandoninck S, Verschaeve L, 1997: Cytogenetic effects of 935.2-MHz (GSM) microwaves alone and in combination with mitomycin C. Mutat Res 393(1-2): 151-156.

Magone, I., 1996: " The effect of electromagnetic radiation from the Skrunda Radio Location Station on *Spirodela polyrhiza (L.) Schleiden* cultures". The Science of the Total Environment, Vol 180, pp 75-80.

Magras, I.N. and Xenos, T.D., 1997: "RF radiation-induced changes in the prenatal development of mice". Bioelectromagnetics 18: 455-461.

Malyapa, R.S., Ahern, E.W., Bi, C., Straube, W/L/., LaRegina, M., Pickard, W.F. and Roti Roti, J.L., 1998: "DNA damage in rat brain cells after in vivo exposure to 2450 MHz electromagnetic radiation and various methods of euthanasia". Radiation Research 149(6): 637-645.

Mann, K.,and Roschkle, J, 1995: "Effects of pulsed high-frequency electromagnetic fields on human sleep". Neuropsychobiology, 33: 41-47.

Mann, K, Roschke, J, 1996: Effects of pulsed high-frequency electromagnetic fields on human sleep. Neuropsychobiology 33(1):41-47.

McKenzie, D.R., Yin, Y. and Morrell, S., 1998: "Childhood incidence of acute lymhoblastic leukaemia and exposure to broadcast radiation in Sydney - a second look". Aust NZ J Pub Health 22 (3): 360-367.

Meltz, M.L., 1995: "Biological effects versus health effects: an investigation of the genotoxicity of microwave radiation". In: Radiofrequency Radiation Standards, NATO ASI Series (B.J. Klauebberg Ed). New York, Plenum Press, 1995: 235-241.

Michelozzi, P., Ancona, C., Fusco, D., Forastiere, F. and Perucci, C.A., 1998: "Risk of leukamia and residence near a radio transmitter in Italy". ISEE/ISEA 1998 Conference, Boston Mass. Paper 354 P., Abstract in Epidemiology 9(4):S111.

Mild, K.H., Oftedal, G., Sandstrom, M., Wilen, J., Tynes, T., Haugsdal, B. and Hauger E., 1998: "Comparison of symptoms by users of analogue and digital mobile phones - A Swedish-Norwegian epidemiological study". National Institute for working life, 1998:23, Umea, Sweden, 84pp.

Milham, S., 1982: "Mortality from leukemia in workers exposed to electric and magnetic fields". New England J. of Med., 307: 249-250.

Milham, S., 1985: "Silent Keys", Lancet 1, 815, 1985.

Milham S., 1985: "Mortality in workers exposed to electromagnetic fields. Environ Health Perspectives 62:297-300.

Milham, S., 1988: "Increased mortality in amateur radio operators due to lymphatic and hematopoietic malignancies". Am. J. Epidemiology, Vol 127, No.1, pp 50-54.

Milham, S., 1996: "Increased incidence of cancer in a cohort of office workers exposed to strong magnetic fields". Am. J. Ind. Med. 30(6): 702-704.

Moscovici, B., Lavyel, A. and Ben Itzhac, D., 1974: "Exposure to electromagnetic radiation among workers". Family Physician 3(3): 121.

Moszczynski, P., Lisiewicz, J., Dmoch, A., Zabinski, Z., Bergier, L., Rucinska, M. and Sasiadek, U., 1999: "The effect of various occupational exposures to microwave radiation on the concentrations of immunoglobulins and T lymphocyte subsets". Wiad Lek 52(1-2):30-34.

Motluk, A., 1997: "Radio head: The brain has its own FM receiver". New Scientist, 25 October 1997, p17.

Naegeli B, Osswald S, Deola M, Burkart F, 1996: "Intermittent pacemaker dysfunction caused by digital mobile telephones". J Am Coll Cardiol 27(6):1471-1477.

Nakamura, H., Seto,T., Nagase, H., Yoshida, M., Dan, S. and Ogina, K., 1997: "Effects of exposure to microwaves on cellular immunity and placental steroids in pregnant rats. Occup Environ Med 54(9):676-680.

Nawrot, P.S., McRee, D.I. and Galvin, M.J., 1985: "Teratogenic biochemical, and histological studies with mice prenatally exposed to 2.45 GHz microwave radiation". Radiation research 102(1):35-45.

Neura, R.R. and del Pizzo, V., 1996: "When 'wire codes' predict cancer better than spot measurements of magnetic fields". Epidemiology 7(3): 217-218.

Nordenson, I., Mild, K.H., Nordstrom, S., Sweins, A. and Birke, E., 1984: "Clastogenic effects in human lymphocytes of power frequency electric fields". Radiat Environ Biophys 23(3): 191-201.

Nordenson, I., Mild, K.H., Ostman, U. and Ljungberg, H., 1988: "Chromosome effects in lymphocytes of 400 kV-substation workers". Radiat Environ Biophys 27(1): 39-47.

Nordenson, I., Mild, K.H., Andersson, G., and Sandstrom, M., 1994: “Chromosomal aberrations in human amniotic cells after intermittent exposure to 50 Hz magnetic fields”. Bioelectromagnetics 15(4):293-301.

Nordstrom, S., Birke, E. and Gustavsson, L., 1983: "Reproductive hazards among workers at high voltage substations". Bioelectromagnetics, 4(1): 91-101.

Occhetta E, Plebani L, Bortnik M, Sacchetti G, Trevi G, 1999: "Implantable cardioverter defibrillators and cellular telephones: is there any interference?". Pacing Clin Electrophysiol 22(7): 983-989.

O'Connor, R.P. and Persinger, M.A., 1997: "Geophysical variables and behavior LXXXII. Strong association between sudden infant death syndrome and increments of global geomagnetic activity - possible support for the melatonin hypothesis". Percept. Mot. Skills, 84(2): 395-402.

ORAU, 1992: "Health Effects of Low-Frequency Electric and Magnetic Fields" Oak Ridge Associated Universities, Washington, U.S.A.

Oscar, K.J. and Hawkins, T.D., 1997: "Microwaves alteration of the blood-brain barrier system of rats". Brain Research 126: 281-293.

Ouellet-Hellstrom, R. and Stewart, W.F., 1993: "Miscarriages among Female Physical Therapists who report using radio- and microwave- frequency electromagnetic radiation." American J. of Epidemiology, 138 (10): 775-86.

Ouellet-Hellstrom, R. and Stewart, W.F., 1995: "Re: Miscarriages among Female Physical Therapists who report using radio- and microwave- frequency electromagnetic radiation." (Reply), American J. of Epidemiology, 141(3), p274.

Penafiel, L.M., Litovitz, T., Krause, D., Desta, A. and Mullins, J.M., 1997: "Role of modulation on the effect of microwaves on ornithine decarboxylase activity in L929 cells". Bioelectromagnetics 18(2): 132-141.

Perry, F.S., Reichmanis, M., Marino, A. and Becker, R.O., 1981: "Environmental power-frequency magnetic fields and suicide". Health Phys 41(2): 267-277.

Persson, B.R.R., Salford, L.G. and Brun, A., 1997: "Blood-brain barrier permeability in rats exposed to electromagnetic fields used in wireless communication". Wireless Network 3: 455-461.

Pfluger, D.M. and Minder, C.E., 1996: "Effects of 16.7 Hz magnetic fields on urinary 6-hydroxymelatonin sulfate excretion of Swiss railway workers". J Pineal Research 21(2): 91-100.

Phelan, A.M., Lange, D.G., Kues, H.A, and Lutty, G.A., 1992: "Modification of membrane fluidity in Melanin-containing cells by low-level microwave radiation". Bioelectromagnetics, 13 : 131-146.

Philips, J.L., Haggren, W., Thomas, W.J., Ishida-Jones, T. and Adey, W.R., 1992: "Magnetic field-induced changes in specific gene transcription". Biochem Biophys Acta 1132(2): 140-144.

Philips, J.L., Haggren, W., Thomas, W.J., Ishida-Jones, T. and Adey, W.R., 1993: "Effect of 72 Hz pulsed magnetic field exposure on ras p21 expression in CCRF-CEM cells". Cancer Biochem Biophys 13(3): 187-193.

Phillips, J.L., Ivaschuk, O., Ishida-Jones, T., Jones, R.A., Campbell-Beachler, M. and Haggnen, W., 1998: "DNA damage in molt-4 T-lymphoblastoid cells exposed to cellular telephone radiofrequency fields in vitro". Bioelectrochem Bioenerg 45: 103-110.

Phillips, J.L., Campbell-Beachler, M., Ivaschuk, O., Ishida-Jones, T., R.A.,. and Haggnen, W., 1998a: "Exposure of molt-4 T-lymphoblastoid cells to a 1g sinusoidal magnetic field at 60 Hz, In: 1998 Annual Review of research on biological effects of electric and magnetic fields from generation, delivery and use of electricity; W/L Associates, Ltd, Frederick, MD.

Polk, C., 1982: "Schumann Resonances". In: CRC Handbook of Atmospherics, Ed: Hans Volland. Boca Raton, Florida: CRC Press, 111-177.

Pollack, H., 1979a: "The microwave syndrome", Bull. N.Y. Acad. Med, 55(11): 1240-1243.

Polson, P. and Merritt, J.H., 1985: "Cancer mortality and Air Force bases. A Re-evaluation". J Bioelectricity 4: 121-127.

Prausnitz, S. and Susskind, C., 1962: “Effects of chronic microwave irradiation on mice”. IRE Trans on Biomed. Elecron. 9: 104-108.

Preece, AW, Iwi, G, Davies-Smith, A, Wesnes, K, Butler, S, Lim, E, Varey, A, 1999: Effect of a 915-MHz simulated mobile phone signal on cognitive function in man. Int J Radiat Biol 75(4):447-456.

Quan, R., Yang, C., Rubinstein, S., Lewiston, N.J., Sunshine, P., Stevenson, D.K. and Kerner, J.A., 1992: "Effects of microwave radiation on anti-infective factors in human milk". Pediatrics 89(4):667-669.

Rao, S. and Henderson, A., 1996: "Regulation of c-fos is affected by electromagnetic fields". J Cell Biochem 63(3): 358-365.

Redelmeier, D.A. and Tibshirani, R.J., 1997: "Association between cellular-telephone calls and motor vehicle collisions". New England J Medicine 336(7): 453-458.

Reiser, H., Dimpfel, W. and Schober, F., 1995: "The influence of electromagnetic fields on human brain activity". Eur. J. Med Res 1(1): 27-32.

Reiter, R.J., 1994: “Melatonin suppression by static and extremely low frequency electromagnetic fields: relationship to the reported increased incidence of cancer”. Reviews on Environmental Health. 10(3-4):171-86, 1994.

Reiter, R.J. and Robinson, J, 1995: "Melatonin: Your body's natural wonder drug". Publ. Bantam Books, New York.

Repacholi, MH, Basten, A, Gebski, V, Noonan, D, Finnie, J, Harris, AW, 1997: Lymphomas in E mu-Pim1 transgenic mice exposed to pulsed 900 MHZ electromagnetic fields. Radiat Res 147(5):631-640.

Robinette, C.D., Silverman, C. and Jablon, S., 1980: “Effects upon health of occupational exposure to microwave radiation (radar)”. American Journal of Epidemiology 112(1): 39-53.

Rosenthal, M. and Obe, G., 1989: "Effects of 50 Hz electromagnetic fields on proliferation and on chromosomal alterations in human peripheral lymphocytes untreated and pretreated with chemical mutagens". Mutation Research 210(2): 329-335.

Rosen, L.A., Barber, I. and Lyle D.B., 1998: "A 0.5 G, 60 HZ magnetic field suppresses melatonin production in pinealocytes". Bioelectromagnetics 19: 123-127.

Rothman, K.J., Chou, C.K., Morgan, R., Balzano, Q., Guy, A.W., Funch, D.P., Preston-Martin, S., Mandel, J, Steffens, R. and Carlo, G., 1996a: "Assessment of cellular telephone and other radio frequency exposure for epidemiologic research. Epidemiology 7:291-298.

Rothman K.J., Loughlin J.E., Funch, D.P. and Dreyer, N.A., 1996b: "Overall mortality of cellular telephone customers". Epidemiology 7:303-305.

Royal Society of New Zealand, 1998: "Radiation and the New Zealand Community". Bulletin No. 34, R.S.N.Z., Turnball Street, Wellington, New Zealand.

Sagripanti, J. and Swicord, M.L., 1976: DNA structural changes caused by microwave radiation. Int. J. of Rad. Bio., 50(1), pp 47-50, 1986.

Salford, L.G., Brun, A., Sturesson, K., Eberhardt, J.L. and Persson, B.R.R., 1994: Permeability of the Blood-Brain Barrier induced by 915 MHz electromagnetic radiation, continuous wave and modulated at 8, 16, 50 and 200 Hz.

Sandyk, R., Anastasiadis, P.G., Annionos, P.A. and Tsagas, N., 1992: "The pineal gland and spontaneous abortions: implications for therapy with melatonin and magnetic field". International Journal of Neuroscience 62(3-4): 243-250.

Sandyk, R. 1993: "Weak magnetic fields antagonize the effects of melatonin on blood glucose levels in Parkinson's Disease". Int. J. Neuroscience 68(1-2): 85-91.

Sandyk, R. 1994: "Rapid normalization of visual evoked potentials by picoTesla range magnetic fields in chronic progressive multiple sclerosis". Int. J. Neuroscience 77(304): 243-259.

Sandyk, R. and Derpapas, K. 1993: "Further observations on the unique efficacy of picoTesla range magnetic fields in Parkinson's Disease". Int. J. Neuroscience 69(1-4): 167-183.

Sandyk, R. and Iacono, R.P., 1993: "Reversal of visual neglect in Parkinson's Disease by treatment with picoTesla range magnetic fields". Int. J. Neuroscience 73(1-2): 93-107.

San Francisco, 1988: "Report on Cancer Incidence in San Francisco". City and County of San Francisco, Department of Public Health, 24 October 1988.

Sanjose, S., Roman, E. and Beral, V., 1991: "Low birthweight and preterm delivery, Scotland, 1981-1984: effect of parent's occupation". Lancet 338(8764): 428-431.

Sarkar, S., Sher, A., and Behari, J., 1994: “Effect of low power microwave on the mouse genome: A direct DNA analysis”. Mutation Research, 320: 141-147.

Sastre, A., Cook, M.R. and Graham, C., 1998: "Nocturnal exposure to intermittent 60 Hz magnetic fields alters human cardiac rhythm". Bioelectromagnetics 19: 98-106.

Savitz, D.A., Wachtel, H., Barnes, F.A., John, E.M. and Tvrdik, J.G., 1988: "Case-control study of childhood cancer and exposure to 60Hz magnetic fields". Am.J. Epidemiology 128: 21-28.

Savitz, D.A., Liao, D., Sastre, A., Klecjner, R.C., and Kavet, R., 1999: "Magnetic field exposure and cardiovascular disease mortality among electric utility workers". Am. J. Epidemiology, 149(2): 135-142.

Savitz, D.A., Checkoway, H. and Loomis, D.P., 1998a: "Magnetic field exposure and neurodegenerative disease mortality among electric utility workers". Epidemiology 9(4):398-404.

Savitz, D.A,, Loomis, D.P. and Tse, C.K., 1998b: "Electrical occupations and neurodegenerative disease: analysis of U.S. mortality data". Arch Environ Health 53(1): 71-74.

Savitz, D.A., Liao, D., Sastre, A., Klecjner, R.C., and Kavet, R., 1999: "Magnetic field exposure and cardiovascular disease mortality among electric utility workers". Am. J. Epidemiology, 149(2): 135-142.

Schlegel RE, Grant FH, Raman S, Reynolds D 1998: "Electromagnetic compatibility study of the in-vitro interaction of wireless phones with cardiac pacemakers". Biomed Instrum Technol 32(6): 645-655.

Schwan, H.P., 1985: "Biophysical principles of the interaction of ELF fields with living matter". Publ. Plenum Press, New York.

Schwartz,, J.L., House, D.E., and Mealing, A.R., 1990: “Exposure of frog hearts to CW or amplitude modulated VHF fields: selective efflux of calcium ions at 16 Hz.” Bioelectromagnetics, 11: 349-358.

Selga, T. and Selga, M., 1996: “Response of *Pinus sylvestris (L.)* needles to electromagnetic fields. Cytological and ultrastructural aspects”. The Science of the Total Environment, Vol 180, pp 65-74.

Selvin, S., Schulman, J. and Merrill, D.W., 1992: "Distance and risk measures for the analysis of spatial data: a study of childhood cancers". Soc. Sci. Med., 34(7):769-777.

Shandala, M.G., Dumanskii, U.D., Rudnev, M.I., Ershova, L.K., and Los I.P., 1979: “Study of Non-ionising Microwave Radiation Effects on the Central Nervous System and Behavior Reactions”. Environmental Health Perspectives, 30:115-121.

Sibbison, J.B., 1990: “USA: Danger from electromagnetic fields”. The Lancet, July 14, 1990, p106.

Sobel, E., Davanipour, Z., Sulkava, R., Erkinjuntti, T., Wikstrom, J., Henderson, V.W., Bucjwalter, G., Bowman, D. and Lee, P-J., 1995: "Occupations with exposure to electromagnetic fields: a possible risk factor for Alzheimer’s Disease". Am J Epidemiol 142(5): 515-524.

Sobel, E., Dunn, M., Davanipour, D.V.M., Qian, M.S. and Chui, M.D., 1996: "Elevated risk of Alzheimer’s disease among workers with likely electromagnetic field exposure. Neurology 47(12): 1477-1481.

Schirmacher, A, Bahr, A, Kullnick, U, Stoegbauer, F, 1999: Electromagnetic fields (1.75 GHz) influence the permeability of the blood-brain barrier in cell culture model. Presented at the Twentieth Annual Meeting of the Bioelectromagnetics Society, St. Pete Beach, FL, June.

Singh, N.P., Stevens, R.E., and Schneider, E.L., 1994: "Modification of alkaline microgel electrophoresis for sensitive detection of DNA damage". Int. J. of Rad. Biolo. 66: 23-28.

Skyberg, K., Hansteen, I.L., and Vistnes, A.I., 1993: “Chromosome aberrations in lymphocytes of high-voltage laboratory cable splicers exposed to electromagnetic fields”. Scandinavian Journal of Work, Environment & Health.19(1):29-34.

Stark, K.D.C., Krebs, T., Altpeter, E., Manz, B., Griol, C. and Abelin, T., 1997: "Absence of chronic effect of exposure to short-wave radio broadcast signal on salivary melatonin concentrations in dairy cattle". J Pineal Research 22: 171-176.

Steneck, N.H., Cook, H.J., Vander, A.J. and Kane, G.L, 1980: "The origins of U.S. safety standards for microwave radiation". Science, 208 (13 June): 1230-1237.

Szmigielski, S., Szudzinski, A, Pietraszek, A., et al., 1982: "Accelerated development of spontaneous and benzopyrene induced skin cancer in mice exposed to 2450 MHz microwave radiation". Bioelectromagnetics, 3: 179-191.

Szmigielski, S., Bielec, M., Lipski, S., and Sokolska, G., 1988: "Immunological and cancer-related aspects of exposure to low level microwave and radiofrequency fields". In Marino (Ed), 'Modern Bioelectricity’, Marcel Bekker, N.Y., pp 861-925.

Szmigielski, S., 1991: International Science Meeting, Beograd, 8-11 April 1991, p 34.

Szmigielski, S., 1996: “Cancer morbidity in subjects occupationally exposed to high frequency (radiofrequency and microwave) electromagnetic radiation”. Science of the Total Environment, Vol 180, 1996, pp 9-17.

Szmigielski, S., Bortkiewicz, A., Gadzicka, E., Zmyslony, M. and Kubacki, R., 1998: "Alteration of diurnal rhythms of blood pressure and heart rate to workers exposed to radiofrequency electromagnetic fields". Blood Press. Monit, 3(6): 323-330.

Taskinen, H., Kyyronen, P., and Hemminki, K., 1990: “Effects of ultrasound, shortwaves and physical exertion on pregnancy outcome in physiotherapists”. J. of Epidemiology and Community Health, 44:196-210.

Thomas, T.L., Stolley, P.D., Stemhagen, A., Fontham, E.T.H., Bleecker, M.L., Stewart, P.A., and Hoover, R.N., 1987: "Brain tumor mortality risk among men with electrical and electronic jobs: A case-control study". J. Nat. Canc. Inst., Vol 79, No.2, pp 233-238., August 1987.

Timchenko, O.I., and Ianchevskaia, N.V., 1995: “The cytogenetic action of electromagnetic fields in the short-wave range”. Psychopharmacology Series, Jul-Aug;(7-8):37-9.

Tonascia, J.A. and Tonascia, S., 1969: "Hematological Study: progress report on SCC 31732", George Washington University, Department of Obstectrics and Gynecology, February 4, 1969.

Tornqvist, S., Knave, B., Ahlbom, A., and Persson, T., 1991: “Incidence of leukaemia and brain tumours in some ‘electrical occupations’”. British Journal of Industrial Medicine, 48: 597-603.

Trigano AJ, Azoulay A, Rochdi M, Campillo, A., 1999: "Electromagnetic interference of external pacemakers by walkie-talkies and digital cellular phones: experimental study. Pacing Clin Electrophysiol 22(4 Pt 1): 588-593.

Tyler, P.E., 1975: “Overview of electromagnetic radiation research: Past, present and future". Annals N.Y. Acad. Sci. 247, February 1975, 6-14.

Tynes, T., Hannevik, M., Anderson, A., Vistnes, A.I. and Haldorsen, T., 1996: “Incidence of breast cancer in Norewegian female radio and telegraph operators”. Cancer causes Control., 7(2): 197-204.

Valjus, J., Norppa, H., Jarventaus, H., Sorsa, M., Nykyri, E., Salomaa, S., Jarvinen, P., and Kajander, J., 1993: “Analysis of chromosomal aberrations, sister chromatid exchanges and micronuclei among power linesmen with long-term exposure to 50-Hz electromagnetic fields”. Radiation & Environmental Biophysics, 32(4): 325-36.

Van Wijngaarden, E., Savitz, D.A., Kleckner, R.C., Dai, J. and Loomis, D., 2000: "Exposure to electromagnetic fields and suicide among electric utility workers: a nested case-control study". Occupational and Environ Medicine, 57: 258-263.

Vaneeck, J., 1998: "Cellular Mechanisms of Melatonin Action". Physiol. Rev. 78: 687-721.

Vaughan, T.L., Daling, J.R. and Starzyk, P.M., 1984: "Fetal death and maternal occupation". J. Occup. Med. 676-678.

Velizarov, S, Raskmark, P, Kwee, S, 1999: The effects of radiofrequency fields on cell proliferation are non-thermal. Bioelectrochem Bioenerg 48(1):177-180.

Verkasalo, P.K., Kaprio, J., Varjonen, J., Romanov, K., Heikkila, K., and Koskenvuo, M., 1997: "Magnetic fields of transmission lines and depression". Am. J. Epidemiology, 146(12): 1037-45.

Verschaeve, L., Slaets, D., Van Gorp, U., Maes, A. and Vanderkom, J., 1994: "In vitro and in vivo genetic effects of microwaves from mobile phone frequencies in human and rat peripheral blood lymphocytes". Proceedings of Cost 244 Meetings on Mobile Communication and Extremely Low Frequency field: Instrumentation and measurements in Bioelectromagnetics Research. Ed. D, Simunic, pp 74-83.

Vignati, M. and Giuliani, L., 1997: “Radiofrequency exposure near high-voltage lines”. Environmental Health Perspectives, 105 (Suppl 6): 1569-1573.

Vijayalaxmi, B.Z., Frei, M.R., Dusch, S.J., Guel, V., Meltz, M.L. and Jauchem, J.R., 1997a: "Frequency of micronuclei in the peripheral blood and bone marrow of cancer-prone mice chronically exposed to 2450 MHz radiofrequency radiation". Radiation Research, 147: 495-500.

Vijayalaxmi, B.Z., Frei, M.R., Dusch, S.J., Guel, V., Meltz, M.L. and Jauchem, J.R., 1997a: "Frequency of micronuclei in the peripheral blood and bone marrow of cancer-prone mice chronically exposed to 2450 MHz radiofrequency radiation - a correction". Radiation Research, 148:

Violanti, J.M., 1998: "Cellular phones and fatal traffic collisions". Accid Anal Prev 30(4): 519-524.

Violanti, J.M. and Marshall, J.R., 1996: "Cellular phones and traffic accidents: an epidemiological approach". Accid Anal Prev 28(2): 265-270.

Von Klitzing, L, 1995: Low-frequency pulsed electromagnetic fields influence EG of man. Phys. Medica 11:77-80.

Walleczek, J., 1992: “Electromagnetic field effects on cells of the immune system: the role of calcium signaling”. FASEB J., 6: 3176-3185.

Wagner, P., Roschke, J., Mann, K., Hiller, W. and Frank, C., 1998: "Human sleep under the influence of pulsed radiofrequency electromagnetic fields: a polysomnographic study using standardized conditions". Bioelectromagnetics 19(3): 199-202.

Wang, S.G. 1989: "5-HT contents change in peripheral blood of workers exposed to microwave and high frequency radiation". Chung Hua Yu Fang I Hsueh Tsa Chih 23(4): 207-210.

Wei, L.X., Goodman, R. and Henderson, A., 1990: "Changes in levels of c0myc and histone H2B following exposure of cells to low-frequency sinusoidal electromagnetic fields: evidence for a window effect". Bioelectromagnetics, 11(4): 269-272.

Wertheimer, N. and Leeper, E.,1979: "Electrical wiring configurations and childhood cancer". Am. J. Epidemiology 109: 273-284.

Wertheimer, N, and Leeper, E., 1986: "Possible effects of electric blankets and heated waterbeds on fetal development". Bioelectromagnetics 7:13-22.

Wever, R., 1970: “The effects of electric fields on the circadian rhythmicity in men”. Life Sci. Space Res., 8: 177-187.

Wever R. 1974, ELF-effects on Human Circadian Rhythms. In: Persinger MA editor. ELF and VLF Electromagnetic Field Effects. New York, Plenum Press. p 101-144.

Weyandt, T.B., Schrader, S.M., Turner, T.W. and Simon, S.D., 1996: "Semen analysis of military personnel associated with military duty assignments". Reprod Toxicol 10(6):521-528.

WHO (1981): "Environmental Health Criteria 16: Radiofrequency and Microwaves". World Health Organization, Geneva.

WHO (1993): "Environmental Health Criteria 137: Electromagnetic fields (300 Hz to 300 GHz) Radiofrequency and Microwaves". World Health Organization, Geneva.

Williams, G.M., 1996: "Comment on 'Acute low-intensity microwave exposure increases DNA single-strand breaks in rat brain cells' "by Henry Lai and Narendra P. Singh. Bioelectromagnetics 17: 165.

Wilson, B.W., Wright, C.W., Morris, J.E., Buschbom, R.L., Brown, D.P., Miller, D.L., Sommers-Flannigan, R. and Anderson, L.E., 1990: "Evidence of an effect of ELF electromagnetic fields on human pineal gland function". J Pineal Research 9(4): 259-269.

Wood, A.W., Armstrong, S.M., Sait, M.L., Devine, L. and Martin, M.J., 1998: "Changes in human plasma melatonin profiles in response to 50 Hz magnetic field exposure". J Pineal Research 25(2): 116-127.

Yaga, K, Reiter, R.J., Manchester, L.C., Nieves, H., Sun, J.H. and Chen, L.D., 1993: "Pineal sensitivity to pulsed magnetic fields changes during the photoperiod. Brain Res Bulletin, 30 (1-2): 153-156.

Youbicier-Simo, BJ, Lebecq, JC, Bastide, M, 1998: Mortality of chicken embryos exposed to EMFs from mobile phones. Presented at the Twentieth Annual Meeting of the Bioelectromagnetics Society, St. Pete Beach, FL, June.

Zaret, M.M., 1977: "Potential hazards of hertzian radiation and tumors. NY State J Med, 146-147.